A Dissertation on

STUDY OF ORGANISMS CULTURED AND THEIR DRUG SENSITIVITY IN PERITONEAL FLUID OF PERFORATION PERITONITIS PATIENTS

Dissertation submitted for the degree of

MASTER OF SURGERY

BRANCH-1 (GENERAL SURGERY) AT

MADRAS MEDICAL COLLEGE, CHENNAI

THE TAMILNADU
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APRIL 2013

CERTIFICATE

This is to certify that the dissertation entitled, "**STUDY OF ORGANISMS CULTURED AND THEIR DRUG SENSITIVITY IN PERITONEAL FLUID OF PERFORATION PERITONITIS PATIENTS"** submitted by **Dr.SREDHARAN.M** to The Tamilnadu Dr.M.G.R medical university, Chennai, in partial fulfillment of the requirements for the award of M.S Degree Branch 1 (General Surgery) is a bonafide research work conducted by him under direct supervision and guidance from the year 2010-2012 at Rajiv Gandhi Government General Hospital, Chennai

Dr. S. DEIVANAYAGAM, M.S.,
Professor and Head of Department,
Department of General Surgery,
Madras Medical College,
Chennai- 600 003

Dr.V. KANAGASABAI, M.D.,
Dean,
Madras Medical College,
Chennai - 600 003.

DECLARATION

I, **Dr. SREDHARAN. M,** certainly declare that this dissertation titled **"STUDY OF ORGANISMS CULTURED AND THEIR DRUG SENSITIVITY IN PERITONEAL FLUID OF PERFORATION PERITONITIS PATIENTS"** represents a genuine work of mine. The contributions of any supervisors to the research are consistent with normal supervisory practice, and are acknowledged. I also affirm that this bonafide work or part of this work was not submitted by me or any others for any award, degree or diploma to any other University board, either in India or abroad. This is submitted to The Tamil Nadu Dr. M.G.R Medical University, Chennai in partial fulfillment of the rules and regulations for the award of Master of Surgery degree Branch 1 (General Surgery).

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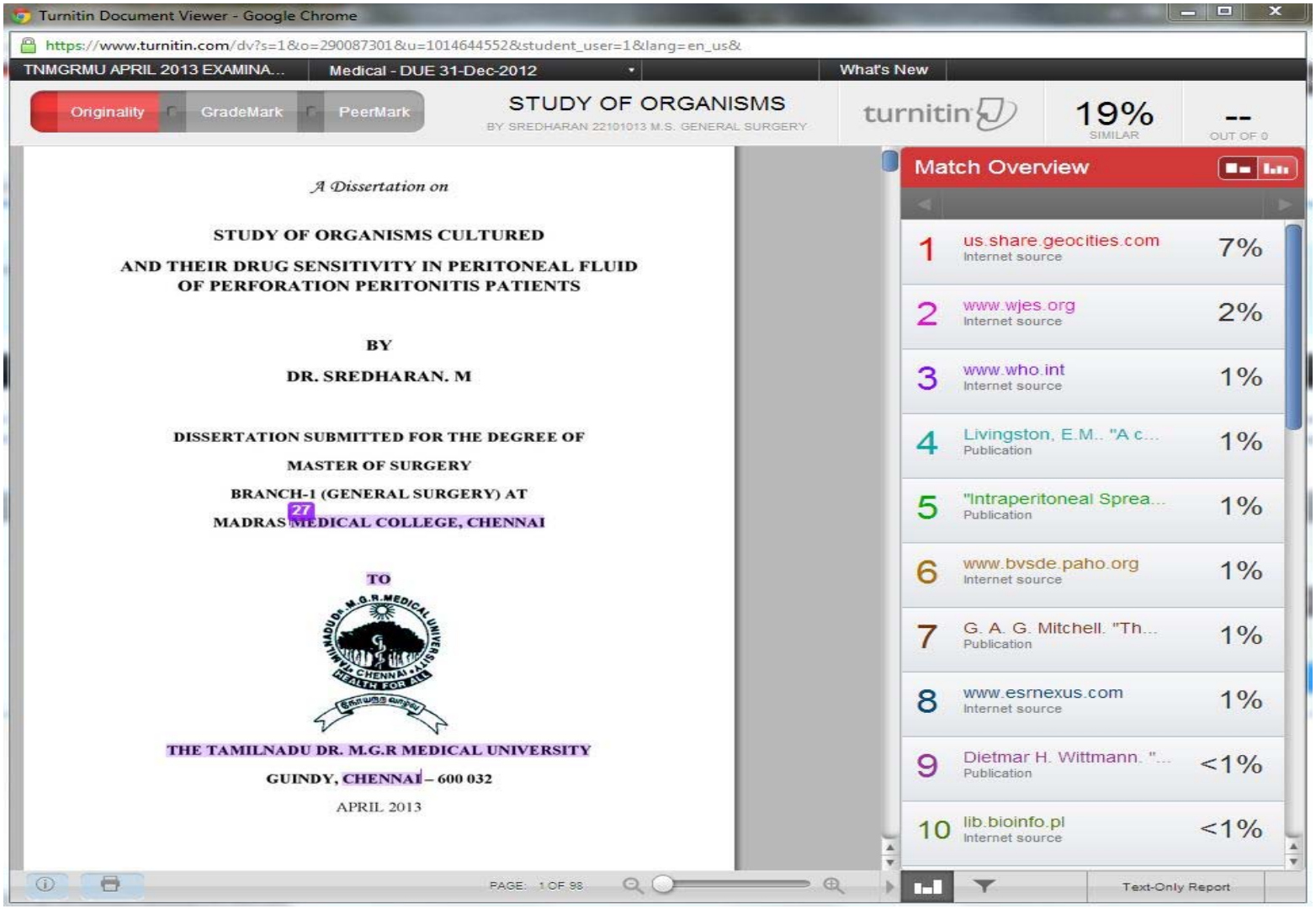
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A Dissertation on STUDY OF ORGANISMS CULTURED AND THEIR DRUG SENSITIVITY IN PERITONEAL FLUID OF PERFORATION PERITONITIS PATIENTS BY DR. SREDHARAN. M DISSERTATION SUBMITTED FOR THE DEGREE OF MASTER OF SURGERY BRANCH-1 (GENERAL SURGERY) AT MADRAS MEDICAL COLLEGE, CHENNAI TO THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY GUINDY, CHENNAI – 600 032 APRIL 2013 CERTIFICATE This is to certify that the dissertation entitled, "STUDY OF ORGANISMS CULTURED AND THEIR DRUG SENSITIVITY IN PERITONEAL FLUID OF PERFORATION PERITONITIS PATIENTS" submitted by Dr. Sredharan. M to The Tamilnadu Dr. M.G.R medical university, Chennai, in partial fulfillment of the requirements for the award of M.S Degree Branch 1...

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CONTENTS

INTRODUCTION

Perforative peritonitis is one of the most common surgical emergencies a surgeon would come across in his career. Among the general surgical conditions it also represents one of the most common causes of mortality. An internship of a surgeon would be termed incomplete had he not come across this ailment at least once.

Perforative peritonitis encompasses a spectrum of disorders that affects the hollow viscera from stomach up to the rectum in its entire length. Even though a myriad of causes lead to the perforation of the bowel, the causes being unique in each part of the bowel, the clinical presentation is disquietingly similar. All these cases present as acute abdomen with tachycardia, abdomen guarding, rigidity, distension and absent bowel sounds. Patients usually are in septicemia with low blood pressure necessitating urgent resuscitation and immediate surgical intervention.

Despite urgent resuscitation and immediate surgical intervention, the death rate among the patients of perforative peritonitis remains high. Septicemia and septic shock leading to sudden cardiac arrest remains the most common cause of death among the perforative peritonitis patients.

One common practice among the leading as well as the budding surgeons, especially in the developing countries like India, is to use the

highest antibiotic available in the hospital or institution in an attempt to reduce the mortality rate. Indiscriminate antibiotic usage is common especially in emergency settings. Though this practice saves some lives in the short term, it leads to evolution multi-drug resistant nosocomial infections in long run, leading to high mortality and cost burden on the patients as well as the state. The debate for using a proper empirical antibiotic continues because of the lack of direct comparative studies in large enough patient samples to make conclusions.

Since the microbacterial spectrum that causes septicemia in perforative peritonitis remains fairly constant it is firmly believed among many surgeons that routine culture and sensitivity of peritoneal fluid wouldn't yield any significant results. Another problem is that the results of the culture sensitivity wouldn't benefit the patient since it takes days to get the results. Nevertheless routine culture sensitivity would simplify the antibiotic regime followed in the institution.

This study is based on the concept that routine antibiotic sensitivity testing and usage of an empirical antibiotic in emergency setting can reduce health care cost by simplifying the antibiotic regime and improve mortality in long run by identifying emergence of drug resistant organisms. This study was conducted with the help of the patients admitted with hollow viscus perforation in Rajiv Gandhi Government General Hospital, Chennai between years 2010-2012.

AIM OF THE STUDY

1. To study the organisms cultured from the peritoneal fluid of patients presenting in the emergency department as acute abdomen and those who have ultrasound evidence of free fluid abdomen by bed side aspiration of the peritoneal fluid.
2. To study the drug sensitivity pattern of the organisms cultured in perforation peritonitis patients.
3. To correlate the organisms cultured to the site of perforation found intra-operatively.
4. To identify the most common drug the organisms are sensitive to.

REVIEW OF LITERATURE

THE PERITONEUM

The earliest descriptions of the peritoneum were found in the Ebers Papyrus of Egypt as early as 1500 B.C. Earlier it was thought as an ordinary covering of the abdomen viscera. The proper anatomical descriptions of peritoneum and omentum and the cavities were done by Douglas and Froriep. It was not until the late 1894, the functions of the peritoneum were known when Staring and Tuby demonstrated the transfer of fluids and other substances from peritoneum to blood and vice versa. These studies were later utilized for the peritoneal dialysis to treat uremia in late 1923 by Ganter.

A proper anatomical knowledge of the peritoneum and its cavities is essential for the surgeon, to understand the development of peritonitis and its evolution in to peritoneal abscess, and to venture in to these spaces during laparotomy for proper drainage.

SURGICAL ANATOMY

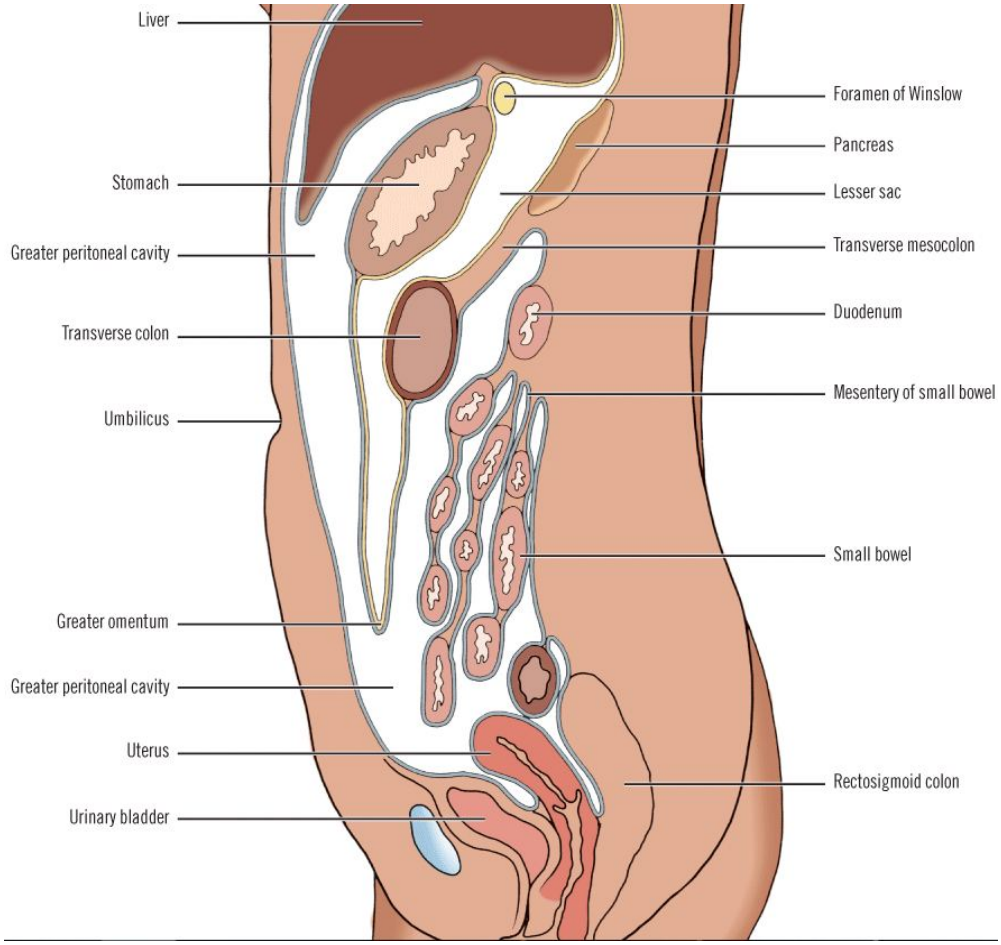
The peritoneum is the largest serous membrane in the body, with a surface area of about 22,000 cm^2. It is divided into parietal and visceral portions. The parietal layer lines the abdominal and pelvic cavities and

the abdominal surface of the diaphragm. The visceral layer covers the abdominal and pelvic viscera including the mesenteries.

The parietal peritoneum is only loosely connected with the body wall, separated from it by an adipose layer, the tela subserosa; whereas the visceral peritoneum is usually tightly attached to the organs it covers. The peritoneum consists of a fibrous layer (the tunica subserosa) and a surface layer of mesothelium (the tunica serosa).

The peritoneal cavity is a potential space. It normally contains only a thin film of fluid which lubricates the surfaces, allowing frictionless movements of the gastrointestinal tract. Under the effects of certain pathologic conditions, great quantities of fluid can occupy the peritoneal cavity.

Peritoneum does not line the entirety of the abdominopelvic cavity. It is lifted from the body wall, especially posteriorly, by organs located against the wall during embryologic development. This chain of events causes the formation of a retroperitoneal space between the peritoneum and the body wall, with organs situated within the space. An organ that is covered only in part by the peritoneum is referred to as a retroperitoneal organ. An organ that is covered by peritoneum essentially everywhere except for the site of entrance of vessels is referred to as an intraperitoneal organ.



VERTICAL DISPOSITION OF PERITONEUM

COMPARTMENTS OF THE PERITONEUM

The peritoneal cavity can be divided into two major compartments by an imaginary cross-sectional plane that passes through the transverse mesocolon. This division defines a supracolic and an infracolic compartment.

Within the supracolic compartment, the liver determines a right and left suprahepatic (subdiaphragmatic) space and a right and left infrahepatic space.

The infracolic compartment is divided by the mesentery of the small bowel into a right infracolic (supramesenteric) compartment, a left infracolic (inframesenteric) compartment, and the pelvic cavity.

The pelvic cavity is divided into right and left spaces by the sigmoid colon and the rectum. It is further subdivided in the female into anterior and posterior spaces by the broad ligament, uterine tubes, and uterus.

SUPRACOLIC COMPARTMENT

SUPRAHEPATIC (SUBPHRENIC) SPACES

Except over the bare area of liver, the serous surfaces of the liver and the diaphragm are in apposition, with a potential space between. This potential space may become the site of intraperitoneal fluid collection and of suprahepatic (subphrenic) abscesses. The potential space is divided into right and left spaces by the falciform ligament.

The right suprahepatic space lies between the diaphragm and the anterosuperior surface of the right lobe and the medial segment of the left

lobe of the liver. The medial boundary is the falciform ligament; the posterior boundary is composed of the right anterior coronary and right triangular ligaments. The inferior boundary is the right lobe and the medial segment of the left lobe of the liver. The space opens into the general peritoneal cavity anteriorly and inferiorly.

The corresponding suprahepatic space on the left (Fig. 10-21) is between the diaphragm and the superior surface of the lateral segment of the left lobe of the liver and the fundus of the stomach. Medially, the left suprahepatic space is bounded by the falciform ligament and, posteriorly, by the left coronary and triangular ligaments. Anteriorly and laterally, the space communicates with the infrahepatic space and the general peritoneal cavity. On the left, the anterior and posterior leaves of the coronary ligament are in apposition.

Localization, extension, and size of the suprahepatic abscess or collection will determine the surgical approach. The surgeon and the radiologist must cooperate to evaluate the anatomy that is altered by the formation of membranes and the pressure of the abscess.

Anteriorly, the approach from beneath the costal margin presents no anatomic complications. Posteriorly, the approach must be by an incision at the level of the spinous process of the first lumbar vertebra to

avoid entering the pleura. Remember the relationship of the pleura and the 12th rib at the vertebral spine. Do not open the bed of the 12th rib.

Nowak et al advocated the dorsolateral approach to the left subphrenic area as well as to the omental bursa and the tail of the pancreas. This is a particularly useful approach by a surgeon or radiologist for the drainage of a left-sided pancreatic abscess complicating acute pancreatitis. The approach does not violate the peritoneal cavity and provides dependent drainage in the recumbent patient.

INFRAHEPATIC SPACES

The right infrahepatic space (subhepatic space, hepatorenal space, pouch of Morison) is bounded superiorly and anteriorly by the right lobe and the medial segment of the left lobe of the liver and the gallbladder, and superiorly and posteriorly by the posterior layer of the coronary and right triangular ligament. Inferiorly, the space opens into the general peritoneal cavity and is partly bounded by the hepatic flexure of the colon and the transverse mesocolon and, medially, by the hepatoduodenal ligament.

The right infrahepatic space communicates with the right suprahepatic space at the:

* Margin of the right lobe of the liver
* Right triangular ligament
* Quadrangular space of Mitchell

The quadrangular space of Mitchell is a small space bounded above by the quadrate lobe of the liver, below by the transverse colon, on the left by the falciform ligament, and on the right by the gallbladder.

Thus the lesser sac of the peritoneum becomes the *left posterior infrahepatic space*. But for practical purposes, the lesser sac or the *omental bursa* are the preferred terms.

INFRACOLIC COMPARTMENT

Below the plane of the transverse mesocolon, the infracolic compartment is divided diagonally by the root of the mesentery of the small intestine. The infracolic compartment is subdivided into a right infracolic (supramesenteric) compartment and a left infracolic (inframesenteric) compartment. Below these areas is the pelvic cavity, and beside them are the paracolic gutters.

The right infracolic space is bounded medially and inferiorly by the root of the mesentery, laterally by the ascending (right) colon, superiorly by the right transverse mesocolon, and anteriorly by the greater omentum. The space is filled with loops of small intestine. It is in communication with the left infracolic space anteriorly and the left pelvic space inferiorly.

The left infracolic space is bounded medially and superiorly by the root of the mesentery, laterally by the descending (left) colon, inferiorly and laterally by the sigmoid colon, and anteriorly by the greater omentum. It communicates with the right infracolic space anteriorly and the right pelvic space inferiorly.

PARACOLIC GUTTERS

There are 2 paracolic gutters. They are located on the lateral and medial sides of the ascending and descending colon.

The right lateral paracolic gutter communicates freely with the right posterior subphrenic space. Therefore, pelvic fluid can reach the diaphragm and fluids from the upper abdomen can drain into the pelvic cavity.Because of the presence of the phrenicocolic ligament, the left

lateral paracolic gutter does not communicate with the perisplenic areas and the left subdiaphragmatic space.

DRAINAGE PATTERNS

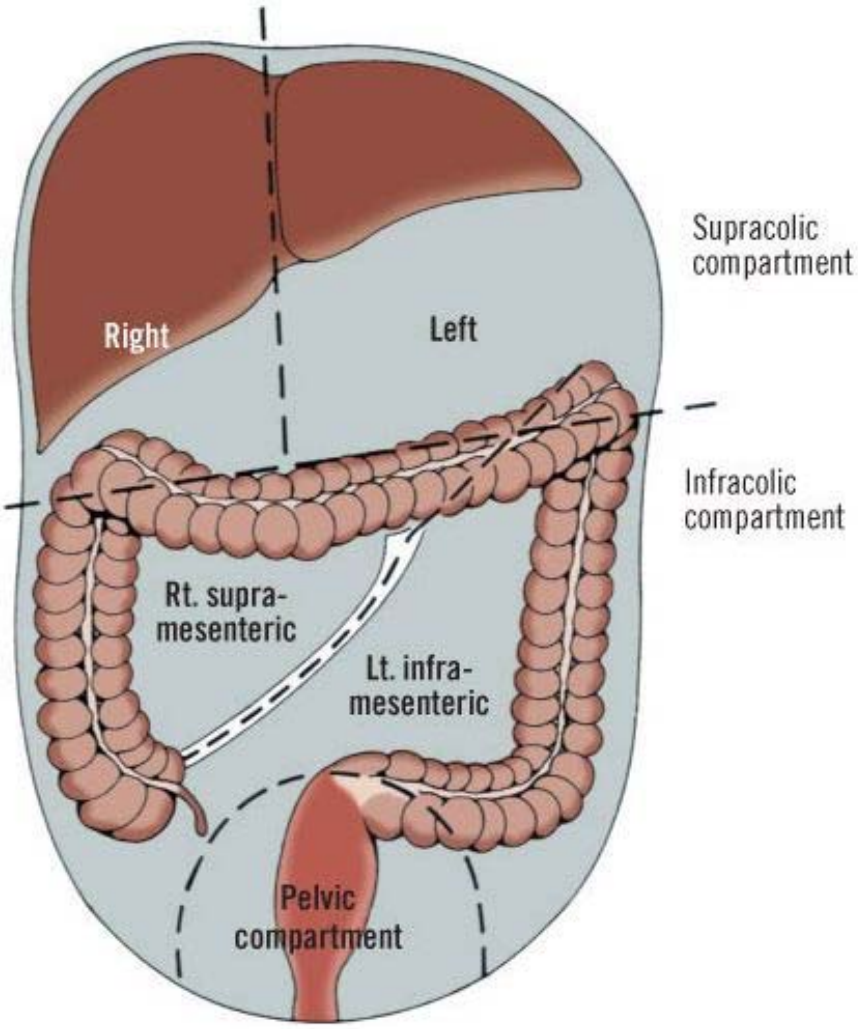
The spread of fluid in the peritoneal cavity depends on all of the following:

* Location of the source and the rate of fluid production
* Pressure differences in the abdomen
* Mesenteric partitions and peritoneal fossae
* Position of the body in relation to gravity

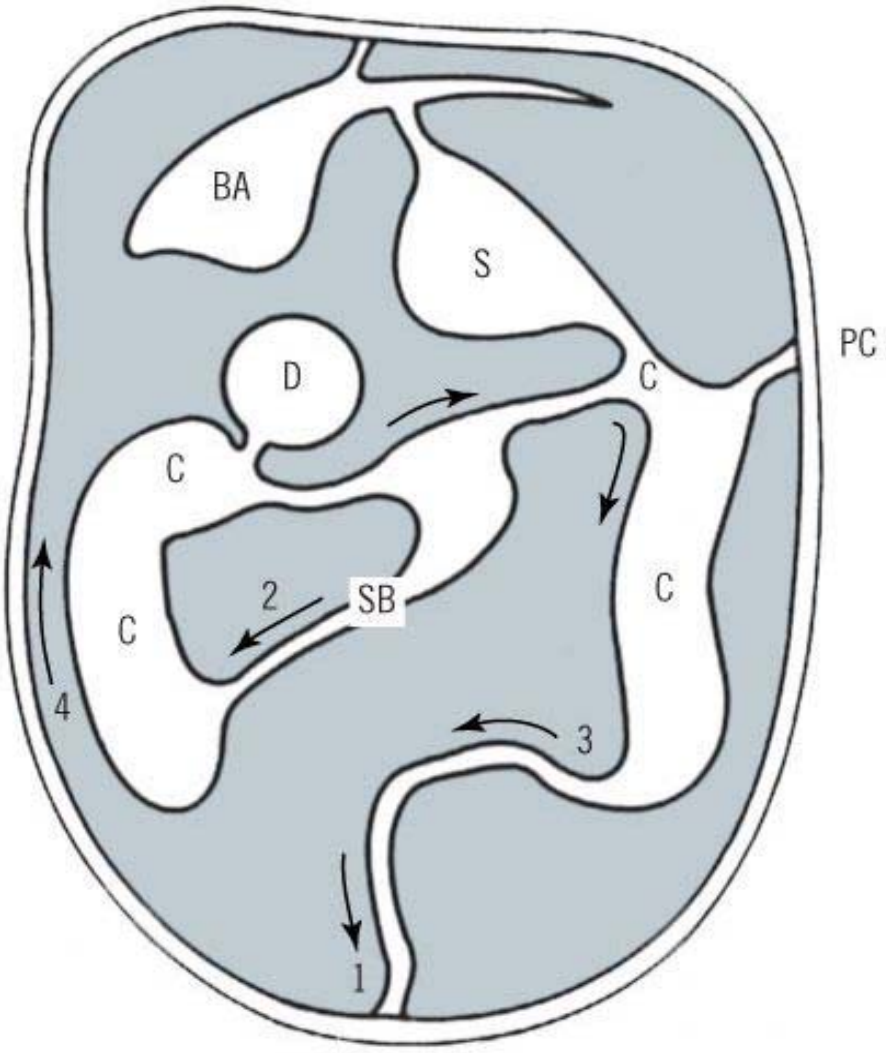
Both infection and metastatic seeding of malignant cells will follow transcoelomic patterns determined by these factors.

PELVIC CAVITY

The sigmoid colon and the rectum divide the pelvic cavity into right and left spaces. In the female, a further division into anterior and posterior spaces is produced by the broad ligament, uterus, uterine tubes, and ovaries. The tunica vaginalis of the male scrotum is embryologically part of the pelvic cavity.



COMPARTMENTALIZATION OF PERITONEAL CAVITY



MESENTERIC ATTACHMENTS AND CHIEF SITES OF SEEDING OF SECONDARIES OF CARCINOMA

1. Pouch of Douglas
2. Distal attachment of mesentery
3. Attachment of sigmoid mesocolon
4. Rt. Para-colic gutter

ANATOMIC SITES OF ABSCESSES

Abscesses and fluid collections may occupy any of the potential spaces. They are usually localized, i.e., walled off by pseudomembranes.

Some of the usual areas of abscesses and collections, discussed earlier in surgical anatomy, are summarized below.

Collections in the Right Anterior Suprahepatic Space

The boundaries are:

- Posterior: Liver
- Anterior: Diaphragm and anterior abdominal wall
- Posteroinferior: Pseudomembranes
- Superior: Pseudomembranes

Collections in the Right Posterior Suprahepatic Space

The boundaries are:

- Superior: Diaphragm
- Anterior: Pseudomembranes and liver
- Inferior: Superior coronary ligament

Collections in the Left Anterior Suprahepatic Space

The boundaries are:

- Right medial: Falciform ligament
- Superior: Left coronary ligament and triangular ligament

- Left inferior: Infrahepatic space
- Posterior: Lateral segment of left hepatic lobe and pseudomembranes
- Anterior: Anterior abdominal wall

Collections in the Left Posterior Suprahepatic Space

The boundaries are:

- Superior: Diaphragm
- Posterior: Left coronary ligament
- Inferior: Liver
- Anterior: Pseudomembranes

Left suprahepatic collections may be walled off by pseudomembranes that bridge the edge of the liver and the abdominal wall. If the collection is larger, the serosa of the stomach and spleen may participate in the sequestration. Pseudomembranes and mesenteric attachments may subdivide the left suprahepatic space into small compartments or collections near the gastric fundus, spleen, or liver.

Collections in the Right Anteroinferior Subhepatic Space

The boundaries are:

- Superior: Liver
- Inferior: Transverse colon and transverse mesocolon
- Anterior and Posterior: Pseudomembranes

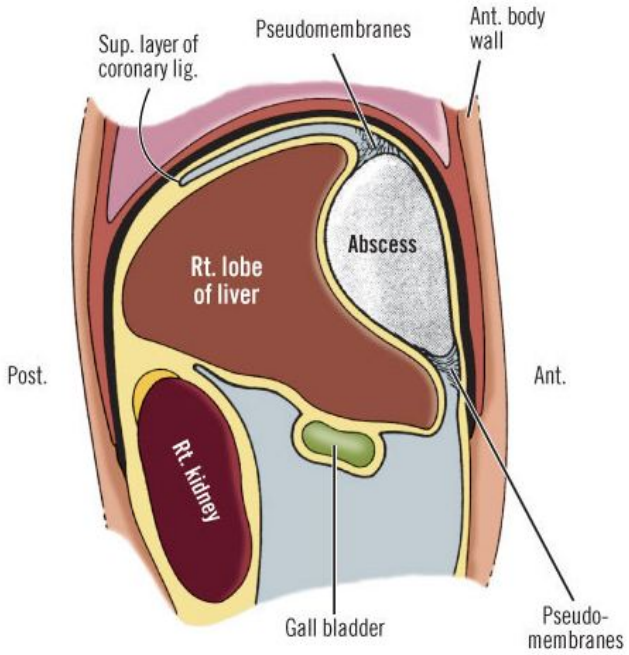
Collections in the Right Postero-superior Subhepatic Space

The boundaries are:

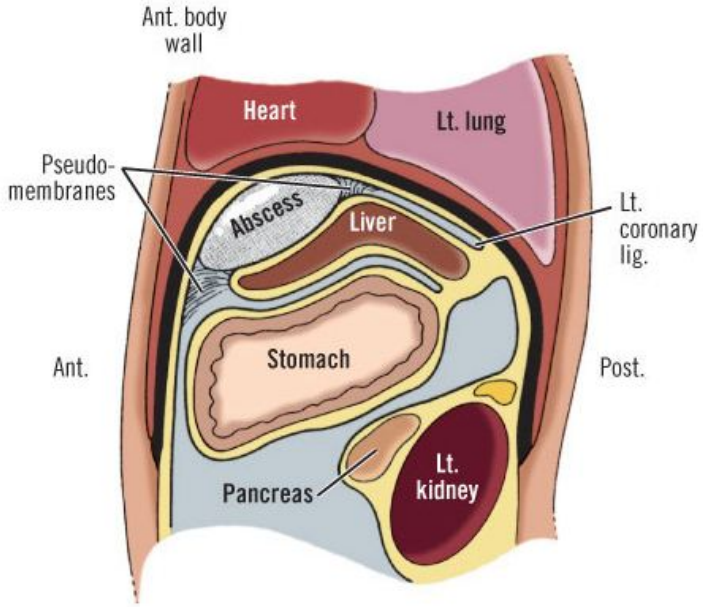
- Superior: Liver
- Posterior: Inferior coronary ligament
- Inferior: Kidney and duodenum
- Anterior: Pseudomembranes

Collections in the Left Anterior Subhepatic Space :

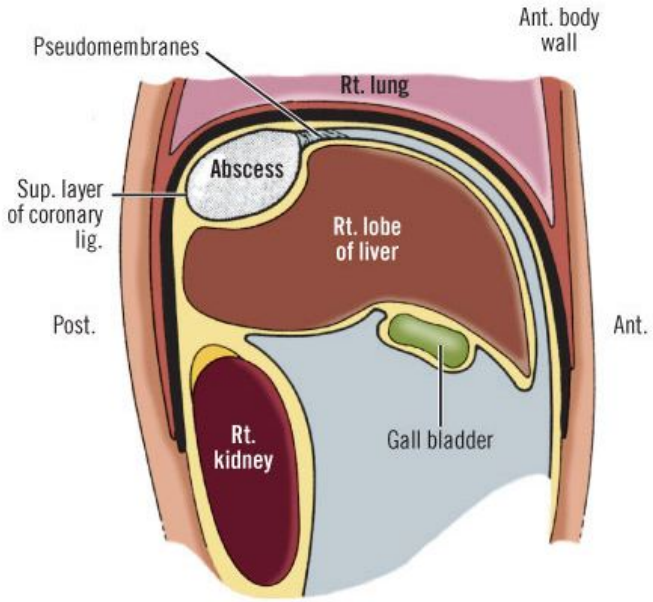
An abscess or fluid collection will probably be limited by the left lateral segment of the liver, the lesser omentum, stomach, and transverse colon. This limitation is not complete. The left segment of the liver is small, so there is free communication with the left suprahepatic space above. Below, the space communicates with the general peritoneal cavity. A typical abscess in this area will be enveloped by adhesions or pseudomembranes between the liver and the anterior abdominal wall and between the abdominal wall and the gastrocolic omentum.



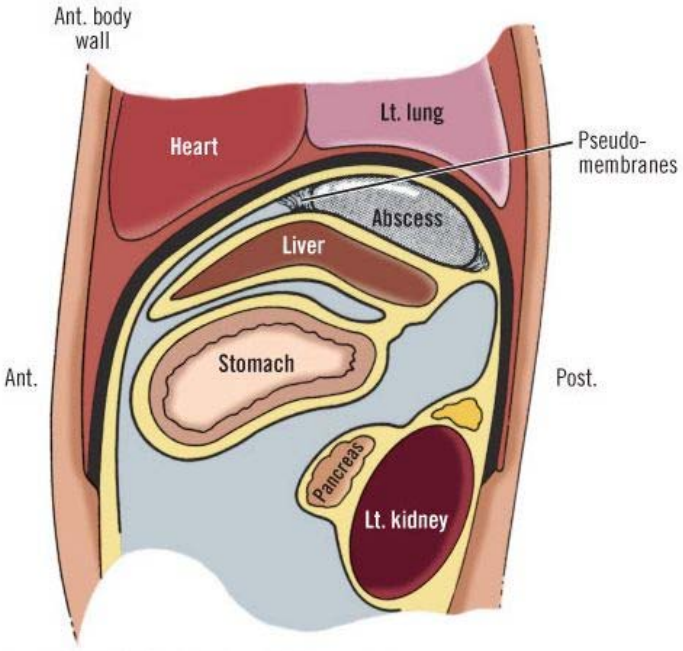
ANTERIOR RIGHT SUPRA-HEPATIC ABSCESS



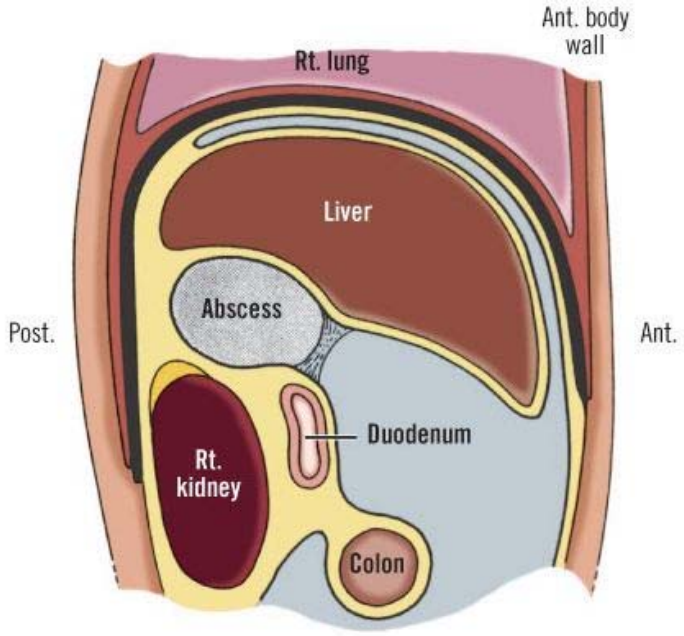
ANTERIOR LEFT SUPRA-HEPATIC ABSCESS



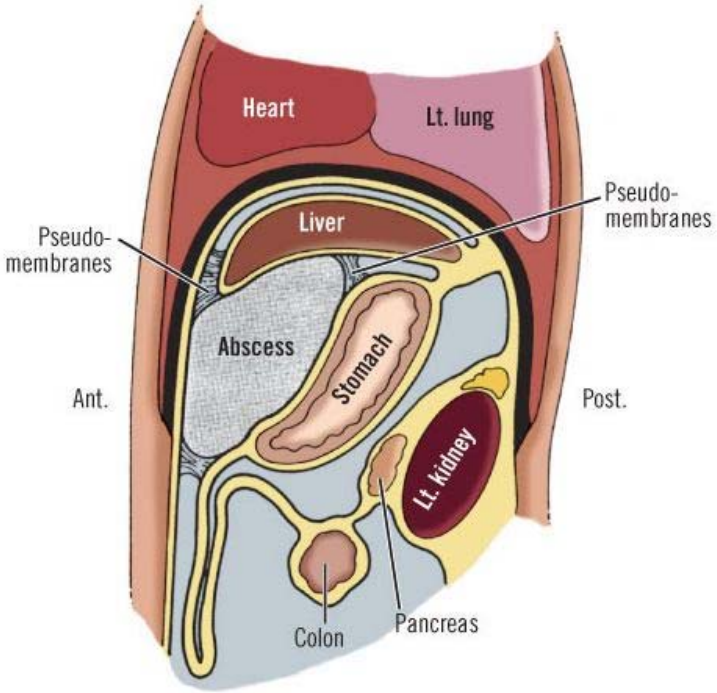
POSTERIOR RIGHT SUPRA-HEPATIC ABSCESS



POSTERIOR LEFT SUPRA-HEPATIC ABSCESS



RIGHT INFRA-HEPATIC ABSCESS



LEFT INFRA-HEPATIC ABSCESS

SURGICAL ANATOMY OF PERITONEUM

In 1932 Livingston very wisely promulgated the following thoughts. Unfortunately, they have been forgotten in an era when embryology and anatomy play a small role in the curriculum of medical students and the training of residents:

Obviously a surgeon possessing clear insight into the modus operandi through which final arrangements in the abdomen are produced is in a position to know how he may safely proceed while operating (in mobilizing portions of bowel; in avoiding important vessels; in relieving intestinal obstruction; in reducing internal hernias; in segregating segments of intestine or creating short-circuits in the alimentary canal); he is qualified to recognize congenital arrests in development when such are encountered. He can differentiate bands and peritoneal folds which represent normal variations, from those which are abnormal formations; and he can judge with reasonable accuracy when intra-abdominal conditions are, and when they are not, consistent with good functional activity. And the diagnostician is aided no less than the operator by a thorough understanding of developmental details.

ADHESIONS

All adhesions (congenital, postoperative, secondary to radiation) should be treated with care. If they are transparent, they should be incised carefully to protect the serosa of the viscus. If they are thick, they should be cut between two clamps, with both parts ligated.

Special attention should be paid to adhesions secondary to radiation enteritis. Morgenstern et al. advised lysis only when absolutely necessary since minute openings into the intestine can be produced, leading to perforation or fistulae. Performing a bypass or a resection is a wise decision.

Shiraishi et al. advised surgical resection of the part of the intestine involved with radiation enterocolitis; this reduces bleeding and improves survival.

PERITONEAL CLOSURE

It is well known that the peritoneum has the capability to heal very rapidly, restoring large defects within hours or days. Differentiated stem cells located within the subperitoneal tissues may be responsible for rapid peritoneal healing. Participation of mesothelial cells in transperitoneal migration and ingrowth may also play a role. Adhesions are formed by a delay in peritoneal healing.

Mazier et al. reported no significant difference in outcome between the primary closure of the pelvic peritoneum and the lack of peritoneal closure. Thome Saint Paul and coworkers asserted that fewer postoperative complications (lymphoceles, fever, etc) developed with visceral nonperitonealization. Spernol et al. strongly advocated that the pelvic peritoneum be left open. A similar recommendation was made by Nagele and coworkers. Than et al. suggested a similar course following abdominal hysterectomy, in particular, Wertheim-Meigs operations. However, Dimpfl and colleagues reported that their own results with 343 women did not give sufficient proof that nonperitoneal closure significantly reduces the incidence of symptomatic lymphocysts.

When treating rectal carcinoma, Chen et al. performed pelvic peritoneal reconstitution to prevent postoperative radiation enteritis. They used the posterior rectus sheath and the peritoneum to partition the abdominal cavity at the umbilical and sacral promontory levels.

EXPLORATION OF THE ABDOMEN

To determine the limits of known disease and to discover unknown or suspected disease, it may be necessary to explore the patient's abdomen. Not every patient with an abdominal complaint can be or should be completely explored. The incision may be too small (as for

appendicitis), there may be extensive adhesions, the patient may be too ill, or the surgeon may encounter peritoneal pus or fluid collections that contraindicate further exploration until the affected areas have been drained or otherwise rendered sterile. Common sense will dictate when to explore and when not to explore.

The diagnosis and treatment of peritonitis, a well-known indication for surgery, is controversial. Chen et al. found ultrasonography a more sensitive technique than clinical judgment in diagnosing peritonitis, especially when the clinical cause was unclear.

Seiler et al. advocated a conservative surgical treatment for diffuse peritonitis which included early intervention, source control, and extensive intraoperative lavage to reduce the reoperation rate.

Bosscha and colleagues gave measured support for open management of peritonitis:

Despite open management of the abdomen and planned re-operation, mortality of severe bacterial peritonitis still continues to be too high, and both short and long-term morbidity are appreciable. The value of open management of the abdomen and planned re-operations rests only

on the clinical observation that other conventional surgical treatments of severe bacterial peritonitis often fail.

The surgeon's approach to exploration of the abdomen is an individual matter. All that is necessary is some system for evaluating normal and pathologic anatomy. Once the surgeon has established a satisfactory pattern of exploration, it should be followed rigidly.

One rule should always be observed: the organ with the primary lesion for which the abdomen has been opened should be left to the end, so that the surgeon's interest in it will not detract from the attention given to the other organs.

Bassett wrote an excellent small book on exploration of the abdomen and the maneuvers necessary to carry it out. He suggested three sequences that may be used: regional, systemic, and circular. The following sequence is drawn from Bassett's regional route:

1. Inspect the abdomen. Note any obvious pathology that (a) may need immediate treatment (i.e., a ruptured spleen) or (b) may contraindicate further exploration (i.e., a perforated colonic diverticular abscess).

2. If the clinical status of the patient warrants further exploration, examine the transverse colon. Pull the colon downward. Examine the supracolic region from right to left. Include the:

- right kidney and adrenal gland
- epiploic foramen of Winslow and common bile duct
- gallbladder
- right lobe of liver
- first and second portions of duodenum
- pancreas
- left lobe segments of liver
- pylorus and lesser curvature of stomach
- fundus of stomach and abdominal esophagus
- greater curvature of stomach
- spleen
- left adrenal area

Pull the transverse colon upward. Examine the infracolic region from right to left. Include the:

- cecum and appendix
- right colon and hepatic flexure

- right retroperitoneal space and lower pole of right kidney
- third and fourth portions of duodenum and superior mesenteric artery
- small intestine and mesentery
- tail of pancreas
- left kidney
- aorta and left retroperitoneal space
- left colon and splenic flexure
- sigmoid colon

3. Examine the pelvic cavity. Include the:

- sigmoid colon
- rectum and uterosacral ligaments
- uterus, tubes, and ovaries
- pelvic wall
- inguinal and femoral regions
- iliac vessels

4. Do not forget to examine the greater omentum. Torsion of the omentum will be obvious. Idiopathic infarction, especially at the right lower end, is obvious.

If this or any other system is used habitually, it will become automatic. As Bassett said: "The goal is to achieve gentleness, accuracy, thoroughness, and speed."

Multiple organ dysfunction syndrome (MODS) calls into question the value of relaparotomy for persisting abdominal sepsis. Koperna and Schulz assert that "...aggressive surgical treatment has reached its limit in patients whose source of infection could not be controlled at the initial operation. To improve overall survival the decision to perform a relaparotomy on demand after an initially successful eradication of the source of infection must be made within 48 hr, at least before MODS emerges."

LAPAROSCOPIC PERITONEAL EXPLORATION

Currently laparoscopy is used as a diagnostic procedure for peritoneal exploration. Muensterer et al. reported that laparoscopy with biopsy of peritoneal nodules is a valuable method by which a histologic diagnosis is established. They warned, though, that port sites must be excised to prevent dissemination of intra-abdominal malignancy. Laparoscopic peritoneal exploration continues to evolve and to be very helpful by avoiding laparotomy.

Estrada Saiz et al. stated that laparoscopy is also useful in diagnosing primary peritoneal mesothelioma as a cause of ascites.

Ditmars and Bongard concluded that the use of diagnostic laparoscopy is very valuable for triage of penetrating trauma; it helps the surgeon decide whether to explore or use conservative treatment. A similar conclusion was expressed by Fernando et al.

The role of laparoscopy in trauma is evolving, according to Poole et al., further research into its diagnostic role and therapeutic applications is clearly needed.

Maffei Faccioli et al. stated that laparoscopy and peritoneal wash represent useful tools in the staging of patients with carcinoma of the pancreas.

Diagnosis of endometriosis is possible with laparoscopy. However, Nezhat et al. cautioned that this procedure should be augmented with techniques utilizing careful palpation and visualization.

A study by al Quorain et al. found that laparoscopy saves unnecessary laparotomies, particularly in cases of tuberculous peritonitis, chronic liver disease, and hepatocellular carcinoma.

Faranda et al. advocated laparoscopic treatment of generalized peritonitis due to perforated sigmoid diverticula for its lower morbidity, shorter hospital stay, and avoidance of colostomy. For early and prompt diagnosis and treatment of tuberculous ascites, Ahmad and Ahmed advise the following diagnostic modalities:

1. Mini laparoscopy
2. Sampling of the ascitic fluid for adenosine deaminase activity and polymerase chain reaction
3. Early antituberculin treatment if such a diagnosis is suspected.

PREVENTION OF PERITONITIS – STRATEGY

1. Prior to surgery – Antibiotics, hyperalimentation, cleansing enemas, respiratory toilet
2. At operation – Irrigations, antibiotics, closed suction drainage, delayed skin closure
3. After surgery – Antibiotics, hyperalimentation, respiratory toilet
4. Technique – Good surgery without "breaks"
5. Understanding of the normal and pathological anatomy of the area

ACUTE PERITONITIS

Acute inflammation of the peritoneum occurring due to a variety of causes is known as peritonitis. It can be classified into various types depending on the type of onset and etiology viz. primary, secondary and tertiary or depending upon the spread of infection viz. diffuse or localized.

CLASSIFICATION ACCORDING TO ETIOLOGY	
I. Primary peritonitis	C. Posttraumatic peritonitis
A. Spontaneous peritonitis in children	1. Peritonitis after blunt abdominal trauma
B. Spontaneous peritonitis in adults	2. Peritonitis after penetrating abdominal trauma
C. Peritonitis in patients with CAPD	3. Other forms
D. Tuberculous peritonitis	**III. Tertiary peritonitis**
E. Other forms	A. Peritonitis without pathogens
II. Secondary peritonitis	B. Peritonitis with fungi
A. Acute perforation peritonitis (acute suppurative peritonitis)	C. Peritonitis with low-grade pathogenic bacteria
1. GI tract perforation	**IV. Intra-abdominal abscess**
2. Bowel wall necrosis (intestinal ischemia)	A. Associated with primary peritonitis
3. Pelviperitonitis	B. Associated with secondary peritonitis

4. Other forms	C. Associated with tertiary peritonitis
B. Postoperative peritonitis	
1. Leak of an anastomosis	
2. Leak of a suture line	
3. Stump insufficiency	
4. Other iatrogenic leaks	

The above table was adopted from “Intra-abdominal infections” published in 1991 by Wittmann DH.

- **PRIMARY PERITONITIS**

Here there is no documented evidence of infection in gastrointestinal tract. The infection usually spreads from the lower genitals through fallopian tubes in females, from upper respiratory tract infection or from middle ear in males. It usually occurs in young malnourished female child under the age of 10 years. Co-existing illness such as nephritis is common. The infection is usually monomicrobial and the source is commonly from non-gastrointestinal source mostly extraperitoneal. The spread occurs via hematogenous spread. It is commonly due to pneumococci and can occasionally be due to

streptococci, hemophilus and other gram negative organisms such as E.coli. The child is toxic and severely ill. Septicemia occurs very early. Treatment is laparotomy and peritoneal toileting under cover of broad spectrum antibiotics. Mortality is very high.

- **SECONDARY PERITONITIS**

As the name suggests it is secondary to any bowel or other visceral pathology viz. hollow viscus perforation, appendicitis. It is the most common form of peritonitis and the infection is commonly polymicrobial usually from intraperitoneal source. E.coli is the commonest organism involved. Other bacteria are aerobic and anaerobic streptococci, Clostridium welchii, bacteroides, Staphylococci, Klebsiella, and Salmonella typhi. Coliforms (E.coli, Klebsiella, Enterobacter & Citrobacter) are the organisms commonly isolated.

- **TERTIARY PERITONITIS**

Tertiary peritonitis is defined as persistent or recurrent intra-abdominal infection after an adequate treatment for primary or secondary peritonitis. It occurs after any abdominal surgeries. It is usually severe leading to early SIRS or MODS.

The other classification system is based on the spread of infection.

Classification According to the Spread of Infection
1. Diffuse peritonitis
2. Localized peritonitis
• Intra-abdominal abscess
• Interloop abscess
• Douglas abscess
• Subphrenic abscess
• Retrocolic abscess
• Pancreas abscess
• Other abscesses

BACTERIA CAUSING PERITONITIS

The bacteria that cause peritonitis can occur either from gastro-intestinal tract or can be from non-gastrointestinal sources.

- **BACTERIA FROM GIT**

  E.coli

  Aerobic and anaerobic Streptococci

  Streptococcus fecalis

  Staphyococcus

  Anaerobes (bacteroides)

  Klebsiella

  Clostridium welchii

- **BACTERIA FROM NON-GI SOURCES**

  Gonococcus
  Pneumococcus
  Chlamydia
  Hemolytic streptococci
  Mycobacterium

Most common bacteria during the phase of peritonitis are E.coli and other coliforms and during abscess formation is Bacteroides fragilis. In Primary peritonitis the most common organisms isolated are the Gram positive cocci.

- **MODE OF INFECTION**

  Hollow viscus perforation

  Penetrating or blunt injury abdomen

  Surgery

  Drains

  Dialysis

  Foreign body

  Appendicitis, cholecystitis, diverticulitis

  Bowel strangulation

  Via fallopian tubes

  Hematogenous spread in septicemia

  Transmural spread

  Following uterine perforation/injury during abortion

- **FACTORS AFFECTING THE SPREAD OF INFECTION**

  Rapidity with which the pus is gushed into the peritoneal cavity

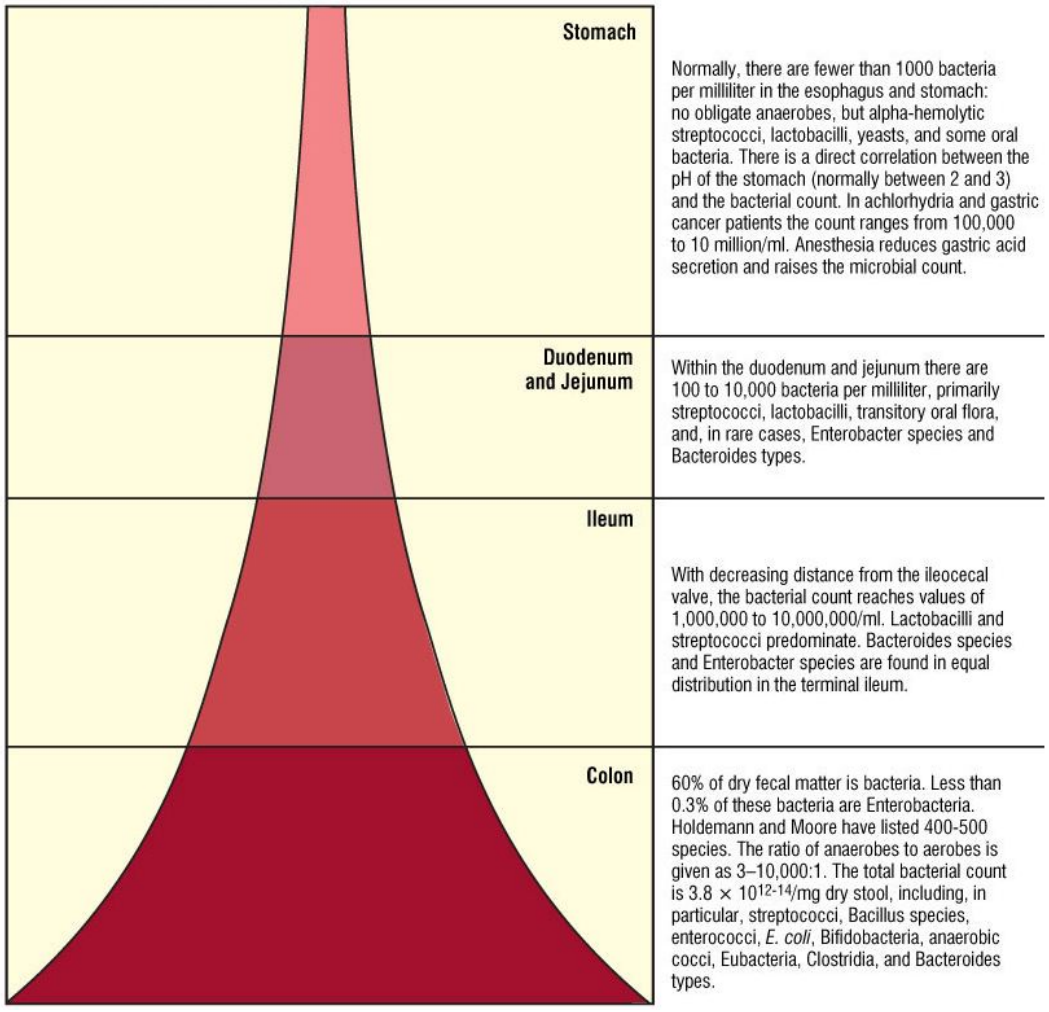
  Amount of peristalsis

  Virulence of organism

  Localizing action and development of the omentum

  Immunosuppression – HIV, steroids

  Host factors viz. malnutrition, anaemia, elderly age



THE VARIATION IN BACTERIAL COUNT IN VARIOUS PARTS OF GIT

PATHOGENESIS OF PERITONITIS

Whenever the peritoneal cavity is infected, protective mechanisms come into action to contain the source of infection. Lots of fluid is secreted into the peritoneal cavity that tends to dilute the toxin. Fibrinogen forms fibrin that localizes the infection. Omentum becomes thickened, edematous and adherent to the site of infection. Bowel loops becomes adherent with each other with fluid collecting between the loops. Thick flakes are formed adhering to the bowel. Peritoneum becomes thick, oedematous, velvety and reddish with loss of its normal glistening appearance due to ongoing inflammation. Often the site of perforation can be identified by the location of the omentum.

Peritoneal contents are initially sterile but eventually become infected. It may either be due to direct spread from the bowel as in perforative peritonitis or due to transmural migration as in pancreatitis. Peritonitis is initially localized due to anatomic factors such as compartmentalization of the peritoneal cavity, greater omentum, dilated small bowel etc. Thickened peritoneum, fibrin deposition, omental adhesions, reduced peristalsis all reduces the spread of infection into the general peritoneal cavity. This leads to localized abscess formation.

Diffuse peritonitis sets in when there is poor localization, rapid peritoneal contamination, virulent organisms, violent peristalsis, immunodeficiency status and poorly developed omentum as in children.

- **CLINICAL FEATURES**

  Sudden onset severe pain in abdomen

  Fever, vomiting

  Tenderness – initially localized but later becomes diffuse

  Rebound tenderness – Blumberg sign

  Guarding and rigidity

  Dull flanks on percussion

  Tachycardia, tachypnoea

  Abdomen distension

  Absent bowel sounds

  Tenderness on PR examination

  Eventual septicemia, septic shock and Hippocratic facies

- **DIFFERENTIAL DIAGNOSIS**

  Acute pancreatitis

  Intestinal obstruction with gangrene

  Ruptured ectopic pregnancy

  Acute mesenteric ischemia

  Acute pyelonephritis

PEPTIC ULCER PERFORATION

It is the terminology used for perforation of duodenal ulcer or gastric ulcer or stomal ulcer. Perforation is common in duodenal ulcer while mortality is common is gastric ulcer perforation and perforation in elderly.

PERFORATED DUODENAL ULCER

It is common in males between the age group of 35-45 years of age group, but can occur in any age group. Anterior ulcer perforates commonly. In 80% of cases there is a history of chronic duodenal ulcer. In 20% of cases it is silent perforation. Perforation can occur in acute ulcers or in acute presentation of pre-existing chronic ulcer.

- **PREDISPOSING FACTORS**
  - Steroids
  - Analgesics (NSAIDS)
  - Alcohol binge
  - Other drugs such as anti-malarials.

Active ulcers perforate commonly. The clinical picture is described in three stages.

- **STAGE OF PERITONISM**

It is due to leakage of gastric juice into the peritoneal cavity causing chemical peritonitis. This stage usually lasts for about six hours. Usually an adult male, who gives a previous history of peptic ulcer, is suddenly seized with acute burning pain over the epigastrium. On examination there is little change in pulse, respiration and temperature.

Tenderness and muscle guarding are constantly present over the site of perforation i.e. the upper half of right rectus muscle. Great importance should be led on the diagnosis of this condition at this stage as chance of survival the patient gradually declines with passage of time.

Diagnosis in first stage mainly rests on two features viz. onset of pain with a dramatic suddenness in a patient who has a previous history of peptic ulcer and muscle guard over the upper half of right rectus muscle. In the late phase of this stage, the pain may be referred to the right iliac fossa due tracking of fluid down along the right paracolic gutter.

- **STAGE OF REACTION (STAGE OF ILLUSION)**

The irritant fluid becomes diluted with peritoneal exudate. The patient feels comfortable and nothing is more deplorable than the

attending doctor sharing the patient's comfort. Symptoms are no doubt relieved but the signs are still there and should be looked for.

Muscular rigidity continues to be present. The other two new features are obliteration of liver dullness and shifting dullness. Rectal examination may elicit tenderness in recto-vesical or recto-uterine pouch. X-ray abdomen erect shows air under diaphragm in most cases. This phase lasts for about 6hrs.

- **STAGE OF DIFFUSE PERITONITIS**

The patient has a pinched anxious face, sunken eyes and hollow cheeks – the classical Hippocratic facies. The patient is usually in a state of shock. The bacteria migrate from the site of perforation causing diffuse bacterial peritonitis. The patient has a rising pulse rate which is low in volume and tension, persisting vomiting, board like rigidity of abdomen, increasing distension of abdomen all hinting the worsening prognosis of the patient and imminent death. Patient may have oliguria, septicemia and septic shock and Hippocratic facies with Multi-organ dysfunction syndrome.

- **CLINICAL FEATURES**

Patient presents with severe persistent pain in the epigastrium initially, later in the right side of the abdomen as the inflammatory fluid

tracks down the paracolic gutter and later this becomes generalized. Abdomen pain is sudden in onset and is due to irritation of parietal peritoneum by the gastric juice and bile. Pain may radiate to the right scapular region and it becomes more on movements. Tenderness and rebound tenderness (Blumberg sign) are seen all over the abdomen. Fever, vomiting, dehydration and oliguria occurs.

Patient is toxic with tachycardia, tachypnoea and hypotension. Abdomen distension occurs with guarding and rigidity all over the abdomen. Dull note is felt in the flanks because of free fluid. Obliteration of liver dullness occurs as a result of escaping gas under diaphragm. The abdomen is silent because of absent bowel sounds. Tenderness is felt on per rectal examination. Sometimes fluid from the supracolic region trickles down along the paracolic gutter and collects in the right iliac region causing pain and tenderness in the right iliac fossa mimicking acute appendicitis.

- **DIFFERENTIAL DIAGNOSIS**

Differential diagnosis for acute abdomen due to perforated peptic ulcer include

Acute pancreatitis

Acute appendicitis when the pus tracks to RIF

Acute cholecystitis

Ruptured aortic aneurysm

Myocardial infarction

Mesenteric ischemia

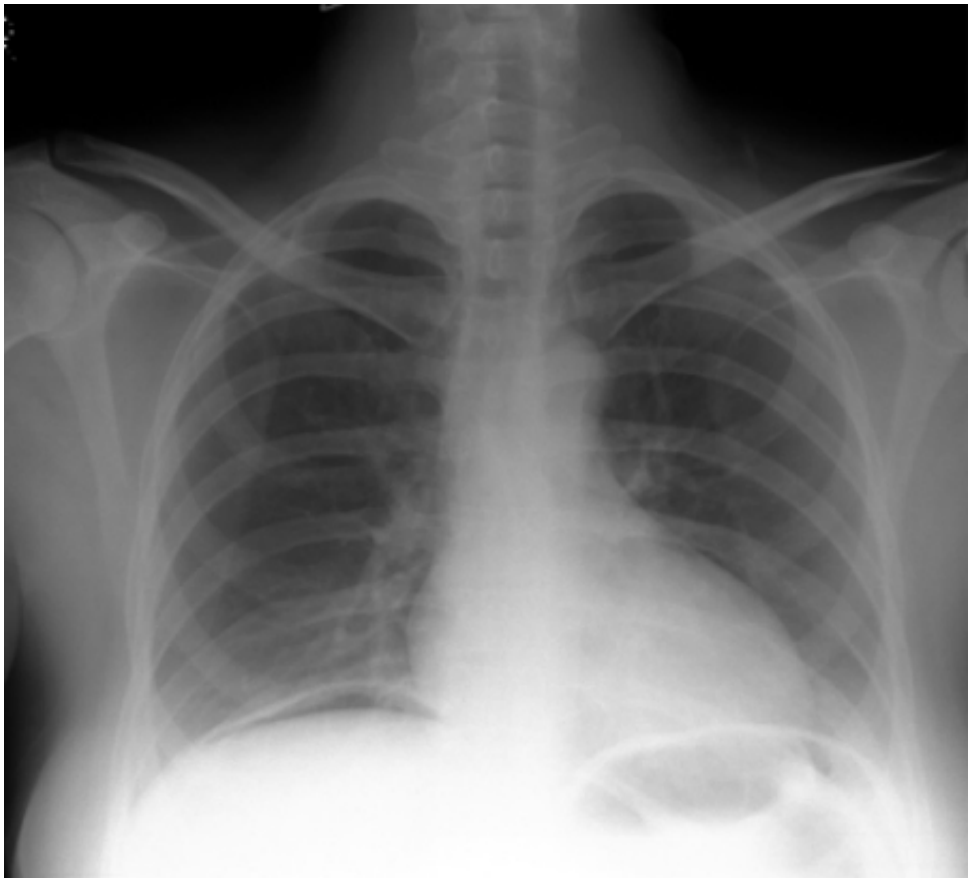
Pneumonia

- **INVESTIGATIONS**

Plain X ray abdomen erect shows gas under diaphragm in >70% cases. In 30% of cases there is no gas under diaphragm because the air leaked is less than 1ml or due to adhesions or due to sealed perforation.

Ultrasound abdomen shows free fluid in abdomen and sometimes gas. CT scan of abdomen is sensitive when there is absent air under diaphragm. It rules out other conditions such as pancreatitis.

Hematological investigations such as blood urea, serum creatinine and electrolytes should be done to assess renal function.



CHEST X RAY PA VIEW ERECT SHOWING AIR UNDER DIAPHRAGM

➢ TREATMENT

Patient is advised admission. Immediate resuscitation is done with intravenous crystalloids viz. dextrose saline, normal saline and ringer lactate. Ryle's tube aspiration is done. Broad spectrum antibiotics are administered. Patient's bladder is catheterized. Patient is shifted to emergency operating room. Laparotomy is done through midline laparotomy incision. All free fluid is sucked out. Perforation site is identified. Thorough peritoneal wash is given with 5-6 liters of luke-warm normal saline. Perforation site is closed with interrupted, horizontal sutures using either silk or vicryl. Omental patch is fixed with loose sutures called the Rosoe-Graham's patch. Drain is placed in the abdomen and abdomen is closed. After recovery patient is kept on proton pump inhibitors for 3months. After 12 weeks a follow up gastro-duodenoscopy is done.

Very rarely in cases of subacute sealed perforations, conservative management is tried with careful observation, intravenous fluids, Ryle's tube aspiration, antibiotics and maintenance of urine output and electrolytes. It is called Hermen-Taylor regime. But this is not a standard treatment. Laparoscopic closure of duodenal ulcer perforation can be done if proper setup is available. It is commonly done for early cases of duodenal ulcer perforation.

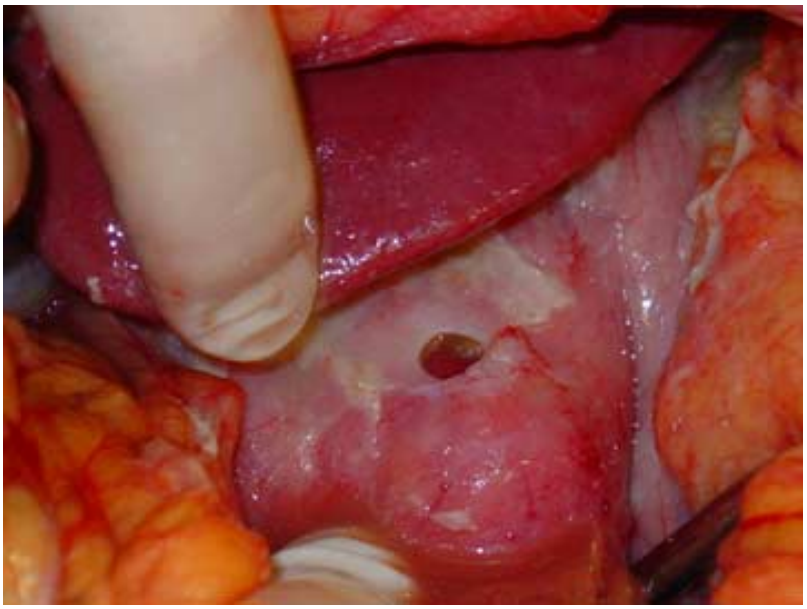
Patient should be prescribed anti-H.pylori therapy for 14days. After that patient should be kept on proton pump inhibitors for 3months. It reduces the chance of reperforation and ulcer recurrence. Manheim's peritonitis index or APACHE II scoring system should be used to assess the patient properly. Occasional large perforated duodenal ulcer with edema that cannot be closed is managed with Roux-en-Y jejunal serosal patch closure with or without feeding jejunostomy.

PERFORATED GASTRIC ULCER

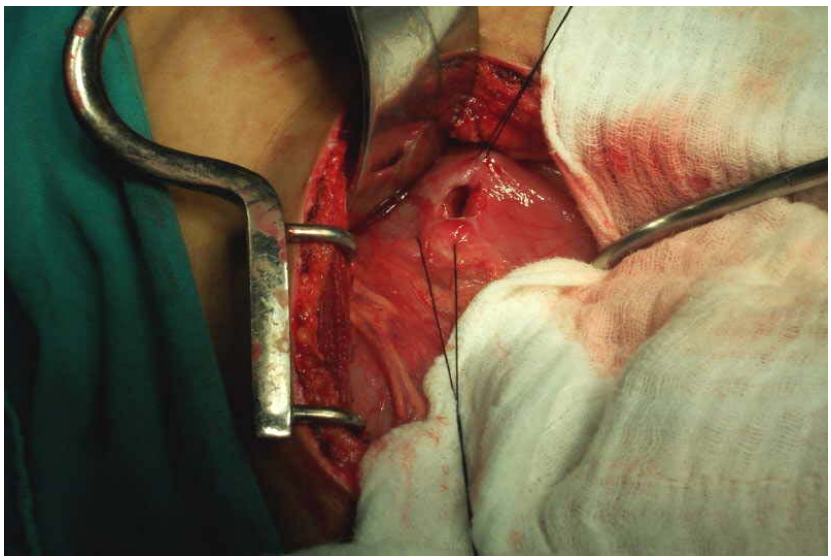
The gastric ulcer that perforates commonly is the lesser curvature ulcer near the antrum. Amount of gas escaped is more than the perforated duodenal ulcer. Malignancy should always be suspected and so biopsy from the edge is a must. Mortality from the gastric ulcer perforation is high when compared to the duodenal ulcer. The immediate resuscitation is similar to that of the duodenal ulcer. Distal gastrectomy including the ulcer area is better option of the patient's general condition is favourable though, primary closure with edge biopsy is the most commonly done procedure.

PERFORATED STOMAL ULCER

Commonly occurs at the gastro-jejunostomy site. The treatment is undoing of the stoma with partial gastrectomy and revision gastro-jejunostomy. Mortality rate is high.



PERFORATED DUODENAL ULCER – INTRA-OPERATIVE



DUODENAL ULCER PERFORATION - CLOSURE

MATERIALS AND METHODS

This is an observational cross section study based on the microorganisms cultured from the peritoneal fluid and their drug sensitivity in patients presenting with free fluid abdomen due to perforative peritonitis in the emergency room. This study was conducted in the period 2011 – 2012, at the Department of General Surgery, Rajiv Gandhi Government General Hospital attached to Madras medical college, Chennai.

After obtaining consent from the ethical committee and informed consent from the patients presenting to the emergency room, patients with free fluid abdomen due to perforative peritonitis were clinically examined and bed side aspiration of peritoneal fluid done and fluid sent to the Microbiology department for culture and drug sensitivity. Only those patients who had an identifiable perforation site in laparotomy and with evidence of free fluid in Ultrasound abdomen were enrolled for this study.

STUDY DESIGN

This study is primarily an observational study and no attempt was made to alter the surgical treatment given to the patients by the treating

surgeon at any point of time. No attempt was made to change the antibiotic regimen preferred by the treating surgeon.

DATA COLLECTION

The following data about the patients are collected and analysed

- Name and sex
- Presenting complaints and its duration
- Presence or absence of fever
- Presence or absence of shock
- Presence or absence of air under diaphragm
- Site of perforation
- Nature of the aspirate
- Antibiotics administered
- Organisms cultured
- Drug sensitivity pattern of the organisms

INCLUSION CRITERIA

1. Patients with peritonitis due to hollow viscus perforation identifiable at laparotomy.
2. Perforative peritonitis patients with evidence of free fluid abdomen in Ultrasound.

EXCLUSION CRITERIA

1. Primary peritonitis patients.
2. Patients with traumatic bowel perforation.
3. Perforative peritonitis patients with no free fluid abdomen.
4. Peritonitis patients with no identifiable perforation site on laparotomy.

DATA ANALYSIS

- Age distribution of perforative peritonitis patients
- Sex wise incidence
- Incidence of various site of perforations
- Incidence of various micro-organisms cultured
- Correlation between duration of symptoms & culture positivity
- Correlation between site of perforation & culture positivity
- Correlation between culture positivity & shock
- Correlation between culture positivity & mortality
- Antibiotic sensitivity pattern among the organisms cultured

PREOPERATIVE WORKUP

The patients presenting to the emergency surgical room at Rajiv Gandhi Government General Hospital, Chennai were clinically evaluated.

A thorough history about the onset of abdomen pain, abdomen distension, constipation, presence or absence of fever and previous history of abdomen pain was taken and documented. Simultaneous resuscitation of the patient with intravenous fluids was done. Ryle's tube was inserted and put under continuous drain. Bladder was catheterized and urine output noted.

The patients were submitted to routine hematological work up with particular importance to serum urea and creatinine. After the patients were adequately resuscitated, they were sent for radiological investigations. Chest X-ray and X-ray abdomen erect were taken. If the patient couldn't stand, lateral recumbent X-ray of the abdomen was taken. Air under diaphragm if any was noted. Ultrasound abdomen was taken and evidence of free fluid was noted. The pre-operative prophylactic antibiotic that was given under the guidance of the assistant surgeon was documented.

After obtaining informed consent from the patient about the procedure, bed side aspiration of the free fluid was done and the nature of the aspirate was noted. The fluid was sent for culture and sensitivity.

The patients were then taken up for emergency laparotomy and the intra-operative findings were noted. The site of perforation and the nature

of contamination were documented. The procedure done was documented. The post-operative antibiotic given was noted and documented.

The patients were then followed up and looked up for surgical site infection, prolongation of sepsis and mortality. Any change in antibiotic during the post-operative period, if any was noted.

Those patients without any evidence of perforation on laparotomy and perforative peritonitis patients without evidence of free fluid abdomen on ultrasound were excluded from the study. Presence or absence of air under diaphragm was documented but not included in inclusion or exclusion criteria.

BED SIDE PERITONEAL FLUID ASPIRATION

EQUIPMENTS REQUIRED

1. Sterile gloves
2. Povidone iodine solution
3. Sterile towel
4. 2% lignocaine solution
5. 10ml syringe
6. 18 gauge iv cannula needle

METHODOLOGY

The bed side aspiration of the peritoneal fluid was done with the patient in supine position. The site of aspiration was painted with 5% povidone iodine solution. The skin was infiltrated with 2% lignocaine after sensitivity testing. Fluid aspiration was done with 18 gauge wide bore intra-venous cannula needle fitted to a 10ml syringe. 8 – 10ml of peritoneal fluid was collected and mixed with the carrier medium, the BACTEC medium in this case.

COMPLICATIONS OF PARACENTESIS AND CULDOCENTESIS

VASCULAR INJURY

Bleeding can result from injury to the superior or inferior epigastric vessels of the abdominal wall or from intraabdominal vessels of the omentum, mesentery, or pelvic region. The result will be a hematoma within the peritoneal cavity or in the abdominal wall, or more grave problems.

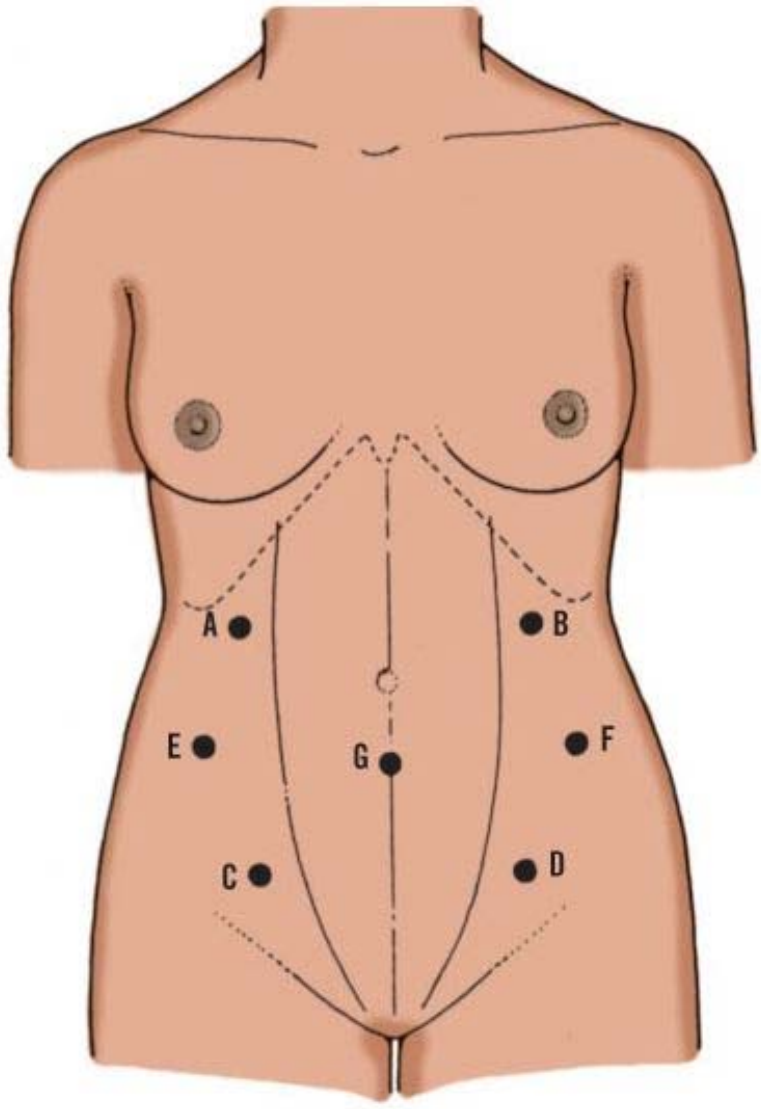
NERVE INJURY

There may be slight discomfort from injury to branches of the 9th to 12th spinal nerves.

ORGAN INJURY

Puncture of the small or large intestine, gallbladder, or urinary bladder is always possible; the bladder should be emptied to minimize the chance of puncture. Intestinal punctures seal quickly. The possibility of sepsis and peritonitis is remote, but the danger is always there.

Injury to the liver during paracentesis is similar to that by liver biopsy. Both bleeding from the spleen and rupture are possible. An ovarian cyst may be injured. Bleeding or even carcinomatosis may result from a ruptured malignant cyst.



POSSIBLE SITES OF PARACENTESIS

- A, B, C, D – Four quadrant aspiration
- E, F – Flank paracentesis
- G – Infra umbilical paracentesis

BED SIDE ASPIRATION OF PERITONEAL FLUID

BACTERIAL CULTURE SENSITIVITY

The peritoneal fluid was sent in sterile test tube containing BACTEC medium to avoid loss of organisms during the transit period. Then the fluid was mixed with various isolation media and then plated to the confirmation media containing the antibiotics routinely used in our hospital.

The various culture media that are commonly used in our hospital and their incubation period are given below.

Medium	Uses	Incubation temperature
Isolation Media		
Lactose broth	Total or thermotolerant coliforms	48 hours at 35 ± 0.5 °C or 37 ± 0.5 °C for total coliforms and 24 hours at 44 ± 0.25 °C or 44.5 ± 0.25 °C for thermotolerant coliforms
MacConkey broth	Total or Thermotolerant coliforms	48 hours at 35 ± 0.5 °C or 37 ± 0.5 °C for total coliforms and 24 hours at 44 ± 0.25 °C or 44.5 ± 0.25 °C for thermotolerant coliforms
Improved formate lactose glutamate medium	Total or Thermotolerant coliforms	48 hours at 35 ± 0.5 °C or 37 ± 0.5 °C for total coliforms and 24 hours at 44 ± 0.25 °C or 44.5 ± 0.25 °C for thermotolerant coliforms

Medium	Uses	Incubation temperature
Lauryl tryptose (lactose) broth	Total or Thermotolerant coliforms	48 hours at 35 ± 0.5 °C or 37 ± 0.5 °C for total coliforms and 24 hours at 44 ± 0.25 °C or 44.5 ± 0.25 °C for thermotolerant coliforms
Confirmatory media		
Brilliant green lactose bile broth	Total or thermotolerant coliforms(gas production)	44.5 ± 0.25 °C for thermotolerant coliforms
EC medium	Thermotolerant coliforms (indole production)	44.5 ± 0.25 °C for thermotolerant coliforms
Tryptone water	Thermotolerant coliforms (gas + indole production)	44.5 ± 0.25 °C for thermotolerant coliforms
Lauryl tryptose mannitol broth with tryptophan	Thermotolerant coliforms (gas + indole production)	44.5 ± 0.25 °C for thermotolerant coliforms

ORGANISMS CULTURED

Common organisms cultured are the following

GRAM POSITIVE

Clostridium species

Corynebacterium species

Enterococcus

Staphylococcus aureus (MRSA & VRSA)

Streptococcus pyrogenes

GRAM NEGATIVE

Bacteroides species

Campylobacter species

Citrobacter species

Escherichia coli

Enterobacter species

Fusobacterium species

Klebsiella species

Prevotella species

Pseudomonas species

Proteus species

Salmonella species

Some of the important organisms involved in peritonitis are discussed below

ESCHERICHIA COLI

- Epidemiology / At Risk:
  - UTI's
  - Gastroenteritis
  - Wound Infections
  - Pneumonia
  - Meningitis in Infants
  - Sepsis
- Manifestations: GI manifestations depend on strain. Virulence factors included.
  - Enteropathogenic E. COLI (EPEC): Cause Travelers's Diarrhea -- loose stools, plus mild GI complaints, such as nausea, vomiting, or even tenesmus. Has also been called Entero-Aggregative E. COLI (EAEC) because of their tendency to aggregate.
  - Enterotoxigenic E. COLI (ETEC): Watery diarrhea, as opposed to loose stools. Diarrhea acts on the small intestine.
    i. Labile Toxin (LT): Heat labile AB-toxin kicks water out by up-regulating cAMP.

ii. Fragment-B: Binds GM1 Ganglioside in small intestinal enterocytes.

iii. Fragment-A: ADP-Ribose Transferase. Transfers ADP to a Gs Stimulatory subunit ------> cAMP.

iv. LT is also a large molecule and a potent antigen.

v. LT-IIa and Iib: Antigenic variants of labile toxin.

vi. Stable Toxin (ST): Heat stable AB-Toxin, prevents water from being reabsorbed in small intestine.

vii. Fragment-A: Up regulated cGMP ------> inhibit reabsorption of Na, Cl, and water in brush border.

- Enteroinvasive E. COLI (EIEC): Causes dysentery in addition to the watery diarrhea. The new cytotoxin acts in the colon, while watery diarrhea continues to occur from the small intestine.

    i. Verotoxin: Phage-mediated Shiga-Like Toxin is cytotoxic to colonic enterocytes. It inactivates protein synthesis at the 60s ribosome and kills the cell, resulting in hemorrhagic necrosis.

    ii. Invasin: Gene allows the E. COLI to live intracellularly inside colonic enterocytes.

- Entero hemolytic E. COLI (EHEC): Causes Hemolytic Uremic Syndrome (HUS). In addition to the dysentery, it has a hemolysin and is tropic for transitional epithelial cells.
    i. Symptoms are
    ii. Hemolytic Crisis
    iii. Thrombocytopenia
    iv. Disseminated Intravascular Coagulopathy (DIC)
    v. Acute Renal Failure
    vi. Hemolysin: Plasmid-mediated factor that lyses red-blood cells. It is also a nephrotoxin

➢ Processing:

- Specimen:
- Stain: Gram-Stain is not done on fecal specimens.
- Culture: Selective Medium (always used on Enterics), which inhibits Gram (+) and contains lactose, in order to differentiate lactose-fermenting genera.

➢ Identification: Lactose-Fermenting

- Motile
- Huge Antigenic Diversity: Flagellar H-Antigen is divided L, A and B subtypes.

i With Neonatal sepsis and meningitis, lab will report a B-subtype as it is associated with prognosis.

ii. The LAB subtypes indicate how easily the flagellar antigens come off with heat.

iii. Mannose-sensitive hemagglutination, because of F1 pilus antigen.

- Virulence: Only cell-associated factors.
  - Pili (F-Antigen): Fimbriae. 10 F Antigen types. Adhesin.
    i. F1 Antigen is found in all E. Coli. (chromosomally coded). It is Mannose-Sensitive, i.e. does not agglutinate RBC's in the presence of mannose (because it prefers to stick to the mannose).

    ii. Mannose sensitive is important to us as normal carriers of E. COLI. It helps E. COLI stick to mucosal (vaginal, GI, buccal) surfaces and protect them.

    iii. F2 - F10 Antigens: They are all mannose-resistant.

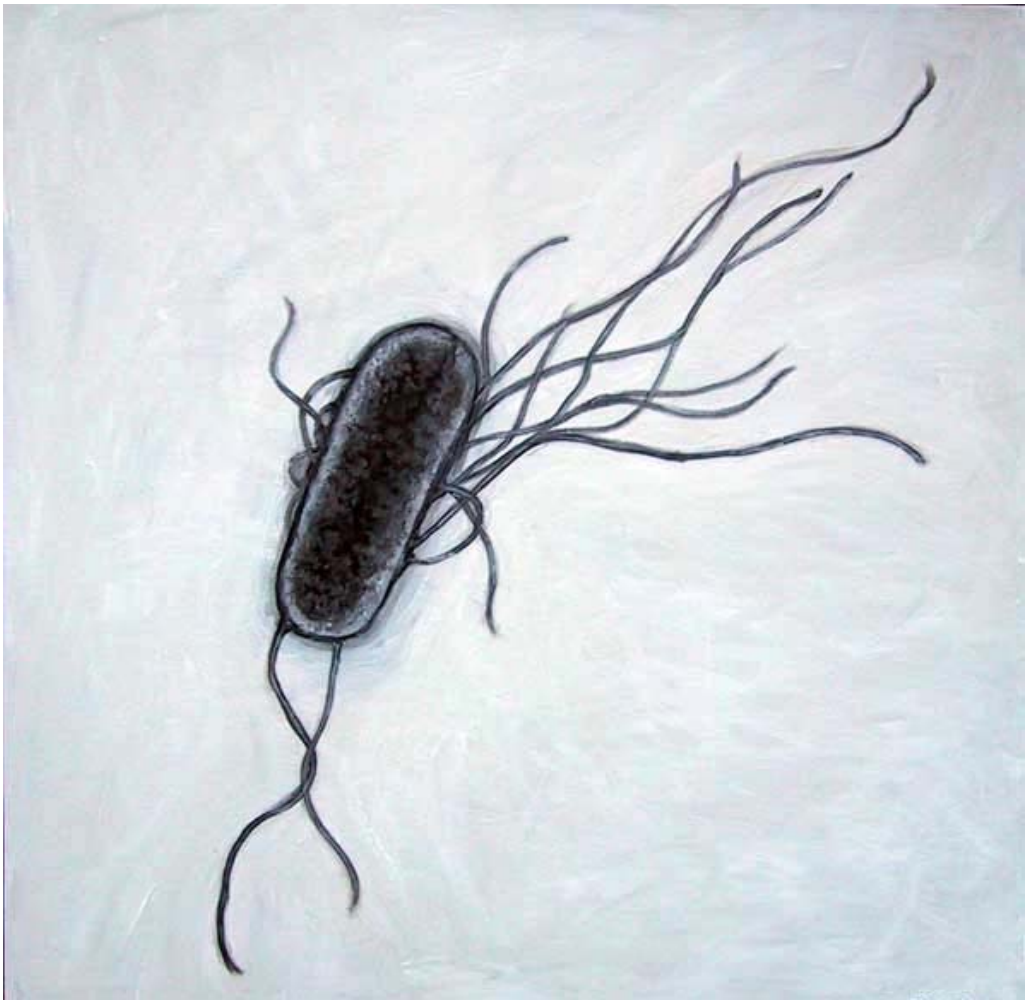
    iv. One of them is the P-Antigen, associated with Pyelonephritis.
  - Capsule (K-Antigen): Important in UTI's and Meningitis. Antiphagocytic, serum resistance.

- Outer Membrane Proteins: Protein-A confers serum resistance.
- Siderophore: Aerobactin
- LPS: Lipid-A is the specific component which is a superantigen and makes up endotoxin.

➢ Vaccine / Prevention:

- The Core Polysaccharide, common to all strands, has been looked at. But whenever we target it, E. COLI respond by making new surface (O-Antigen) polysaccharides.

➢ Treatment: Risk of inducing endotoxic shock when treating bacteremia.



ESCHERICHIA COLI

BACTEROIDES FRAGILIS

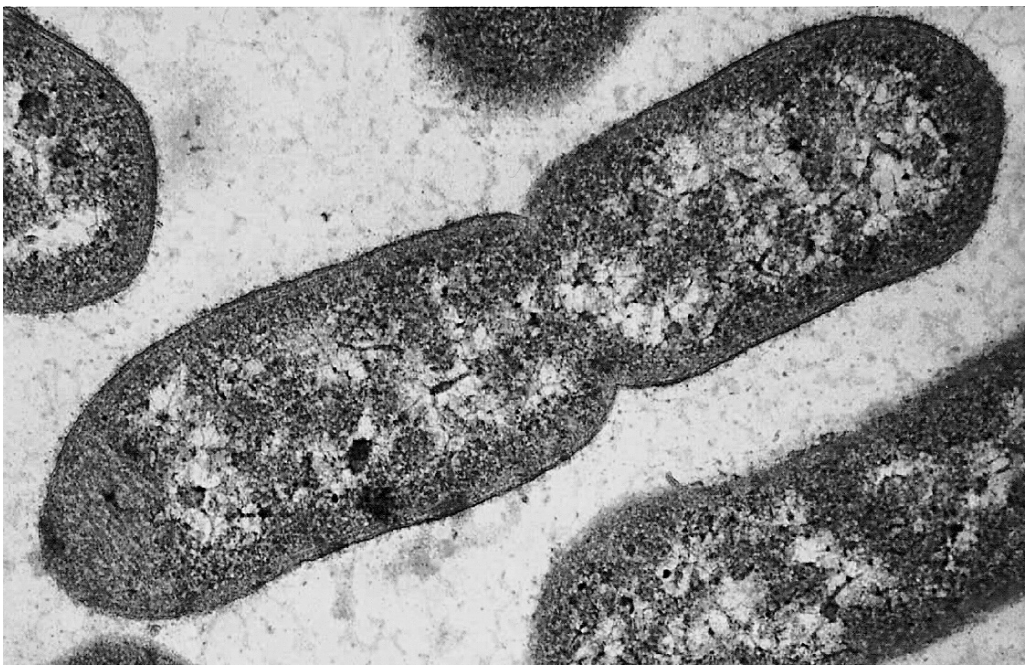
- Gram-Negative Pleomorphic Rod
- Epidemiology / At Risk:
  - #1 bacteria of normal GI flora. 1010-1011 bacteria / gram
  - Can survive on skin and mucosa.
  - At Risk: Immunocompromised
- Manifestations: Bugs enter through a compromise in GI-tract. Initial infection is polymicrobic, but as other bugs use up the oxygen, Bacteroides can grow.
  - Pelvic Inflammatory Disease
  - Intra-abdominal abscess; peritonitis
  - Sustained Bacteremia: Not endotoxic, and not transient, but in between.
- Processing:
  - Specimen: Exudate. Will be polymicrobic early on. Must use anaerobic transport medium.
  - Stain: Gram-negative rods contain intracytoplasmic vacuoles.
  - Culture: Blood agar with antibiotics.
- Identification:
  - Strict (Aerotolerant) Anaerobe

- Direct-FA on capsule to verify its presence. It is not readily visible in culture.
- Gas-Liquid Chromatography of metabolic byproducts. Anaerobes are difficult to culture.

➢ Virulence:

- Isobutyric and Succinic Acid: Biochemical byproducts are toxic to Salmonella and Shigella. Thus our normal flora are protective against Salmonella.
- Capsule: Allows adhesin to peritoneum.
  i. Antichemotactic and antiphagocytic for PMN's.
  ii. Aids to inhibit intracellular killing once the bugs are phagocytosed.
- Superoxide Dismutase: makes it aerotolerant
- Catalase: induced by hemin, thus produced in the blood.
- Enzymes: Hyaluronidase, DNAse, Heparinase contribute to invasiveness.
- LPS: Contains Lipid-A, but it is not as toxic as E. Coli endotoxin. Only mild endotoxic effect.
- Enterotoxin: Diarrhea. This toxin only carried by a few strains.
- beta-Lactamase

➢ Treatment: Abscesses must be surgically drained. Metronidazole.



BACTEROIDES FRAGILIS

ENTEROCOCCUS

- Name-Derivation: feces.
- Epidemiology / At Risk: Normal GI flora.
- Manifestations: Multiple infections. Often a complication of cholecystitis.
  - GI obstruction may lead to bacteremia and endocarditis, due to bacterial resistances.
- Processing:
  - Stain:
    i. GRAM-VARIABLE -- both Gram (+) and Gram (-) found, alive.
  - Culture: Use blood agar with 40% bile and 6.5% NaCl. All three types of hemolysis found in culture.
- Identification:
  - Grows in the presence of bile.
  - Penicillin resistant.
- Virulence:
  - LIPOTECHOIC acid, very lipid rich, leads to gram-variable appearance.
- Treatment: Penicillin resistant, strongly, due to altered Penicillin-binding proteins.
  - Also have acquired vancomycin and gentamycin resistance.

KLEBSIELLA SP.

- Gram (-) Rod.
- Name-Derivation: Klebs was a researcher.
- Epidemiology / At Risk: Male, malnourished, alcoholic pneumonia. "VA pneumonia."
  - Transient normal flora of throat.
- Manifestations:
  - Pneumonia: Acute lobar pneumonia with highly productive cough.
    i. Productive cough. Thick, mucinous, messy, bloody sputum.
    ii. Focal lung abscesses in late infection lead to bacteremia.
  - Bacteremia with secondary Meningitis
  - Wound Infections
  - UTI
- Processing:
  - Culture:
    i. Blood Agar: Difficult to define boundaries because the bugs are so mucinous.
    ii. Selective Medium: Used to inhibit Gram (+) growth; will yield smooth and mucinous colonies.

- Identification:
  - Lactose (+)
  - Serotypes: 77
- Virulence:
  - Capsule: THICK capsule is antiphagocytic.
    i. Confers serum resistance ------> bacteremia.
  - beta-Lactamase
  - Enterotoxin: Plasmid-mediated. Causes vomiting and diarrhea.
  - Endotoxin: LPS, fever, hypotension.
- Treatment:
  - Penicillin-Resistant. Use broad-spectrum cephalosporins.

ANTIBIOTICS TESTED

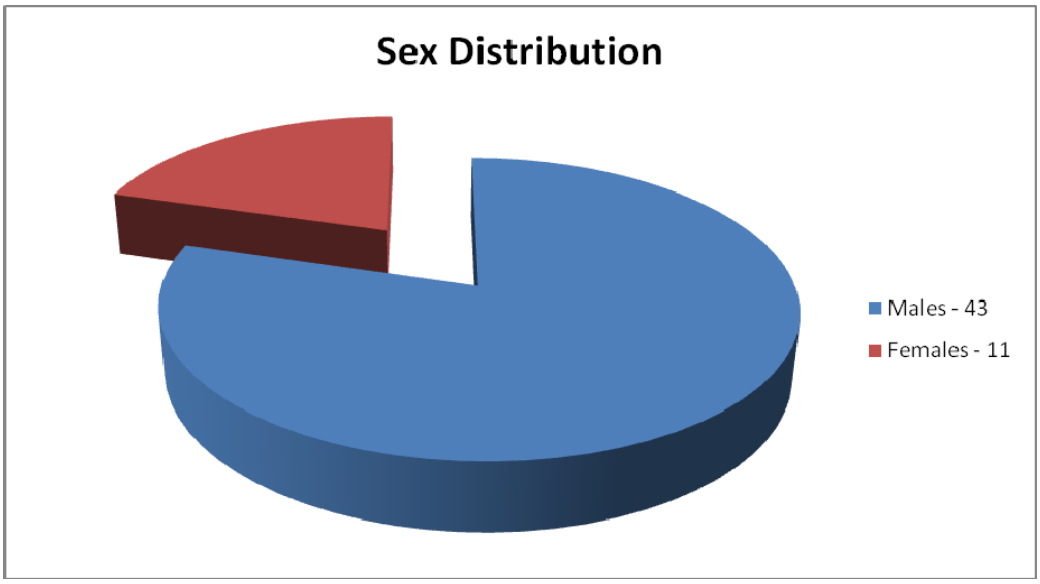
- Amikacin
- Ampicillin
- Cefotaxim sodium
- Ceftriaxone sodium
- Cefoperazone
- Cefoperazone sulbactam
- Ciprofloxacin
- Clindamycin
- Cotrimoxazole
- Crystalline penicillin
- Cloxacillin
- Imipenam
- Meropenam
- Metronidazole
- Ofloxacin
- Piperacillin tazobactam
- Vancomycin

Sensitivity of the organisms for the above mentioned antibiotics is noted as these are the common antibiotics available and used in our hospital.

RESULTS

SEX DISTRIBUTION

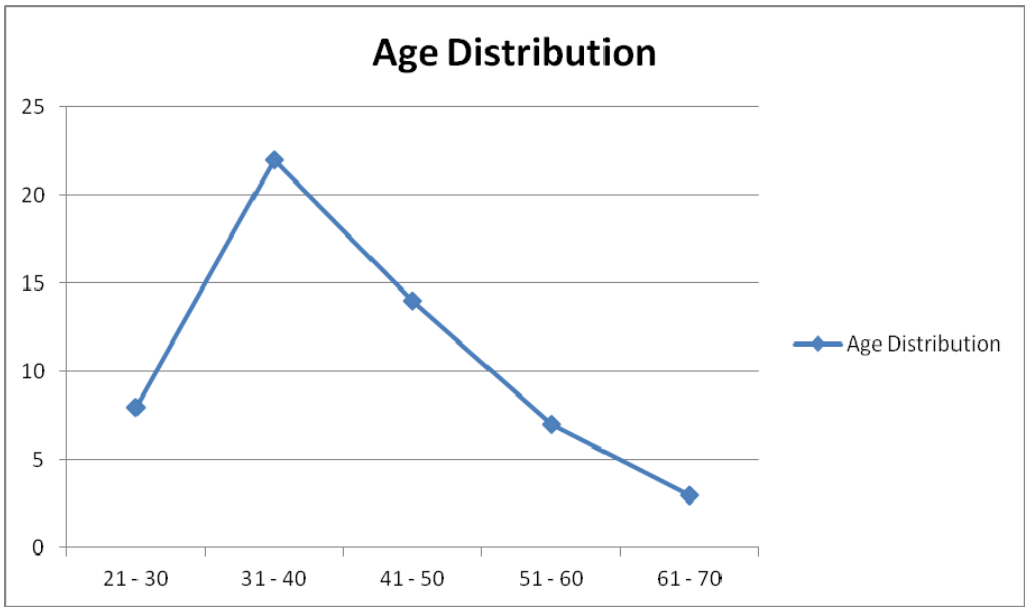
Males are more commonly affected than the females. Out of 54 patients 43 are males (80%) and 11 are females (20%)



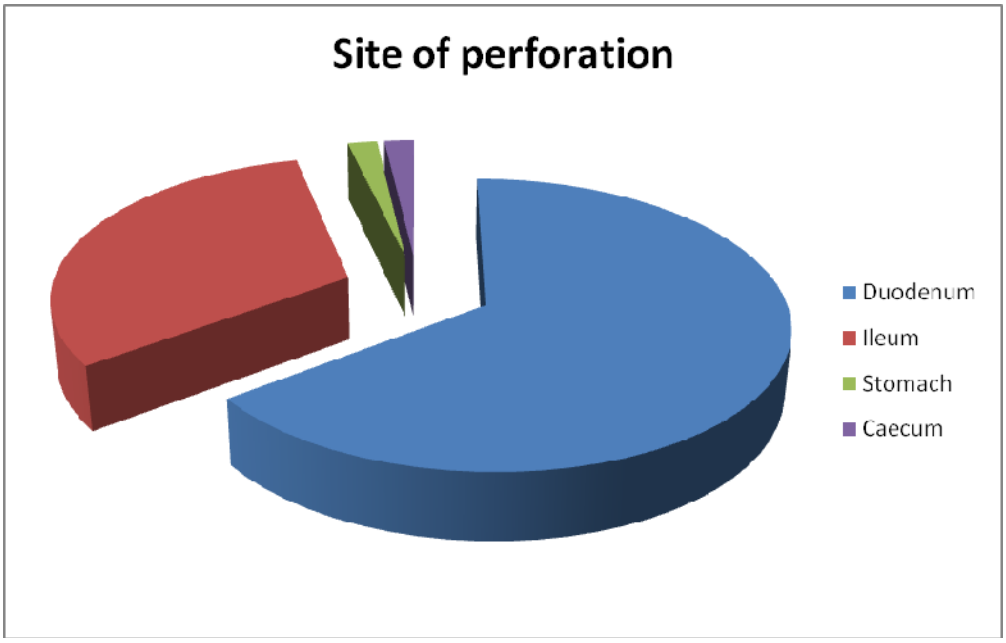
AGE DISTRIBUTION

S. NO.	AGE GROUP	TOTAL
1	21 – 30	8
2	31 – 40	22
3	41 – 50	14
4	51 – 60	7
5	61 – 70	3

Most patients affected by perforative peritonitis belong to the age group of 30-40. It coincides with the age group where the peptic ulcer disease is more prevalent.



SITE OF PERFORATION



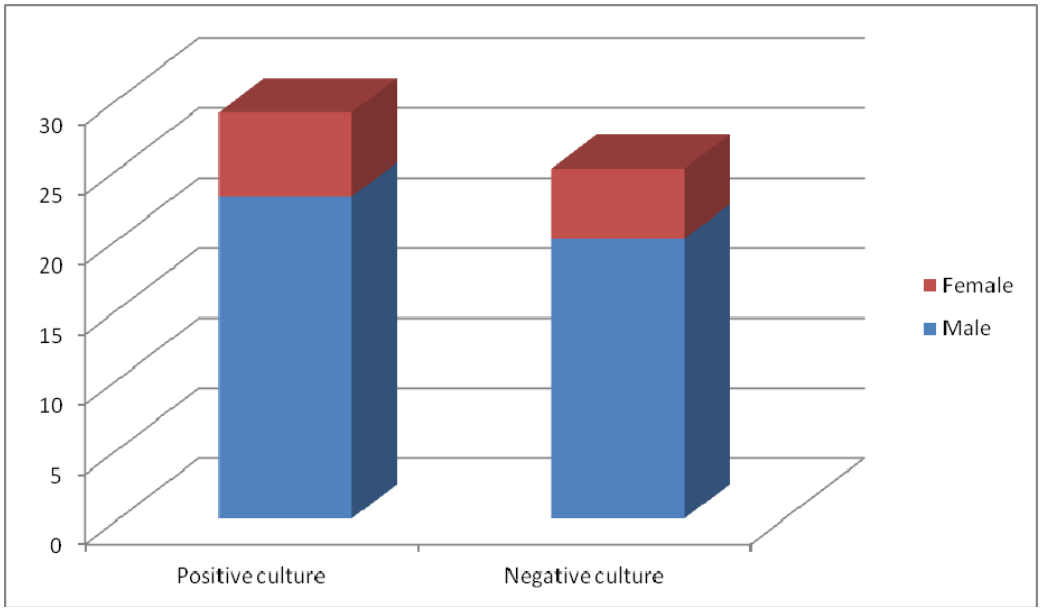
Duodenal ulcer perforation is the commonest cause of perforative peritonitis in the group studied constituting around 65% of all hollow viscus perforations. It is followed by ileal perforation which constitutes 31.5% of cases which is higher than the western counterparts probably because of early detection.

ORGANISMS CULTURED

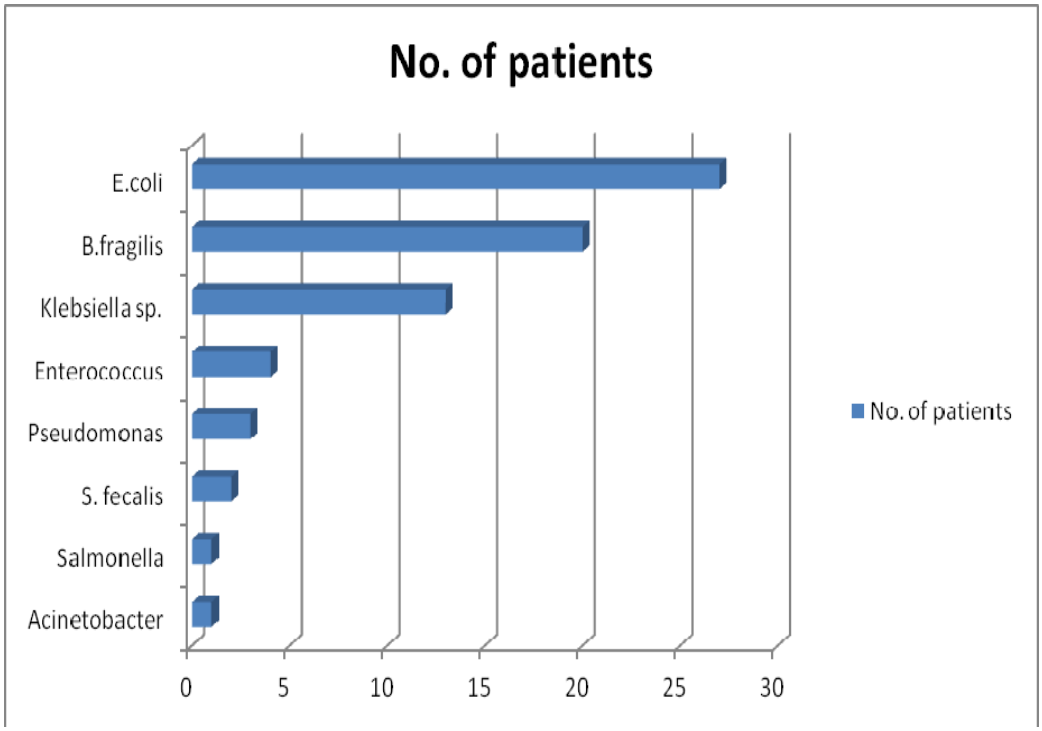
Out of all the 54 peritoneal fluids sent for culture sensitivity, 29 yielded bacterial growth which is around 53.7%. Gram negative enteric bacteriae predominate the culture isolates. Escherichia coli are the most common bacteriae isolated from the peritonitis fluid. It is isolated from 27 of all 54 cultures (50%) and from 29 of positive cultures (93%). It is then followed by gram negative, capsulated, anaerobe Bacteroides fragilis which is isolated from 20 cases of all 54 cultures (37%) and from 29 positive cultures (69%). The other common bacteriae isolated are Klebsiella species (44.8%), Pseudomonas, Streptococcus fecalis and Acinetobacter. The culture negativity remains equal among both male and female patients (46.5% and 45.5% respectively).

GROUP	MALE	FEMALE	NUMBER
POSITIVE CULTURE	23	6	29
NEGATIVE CULTURE	20	5	25
TOTAL	43	11	54

The culture negativity remains equal among both male and female patients (46.5% and 45.5% respectively).

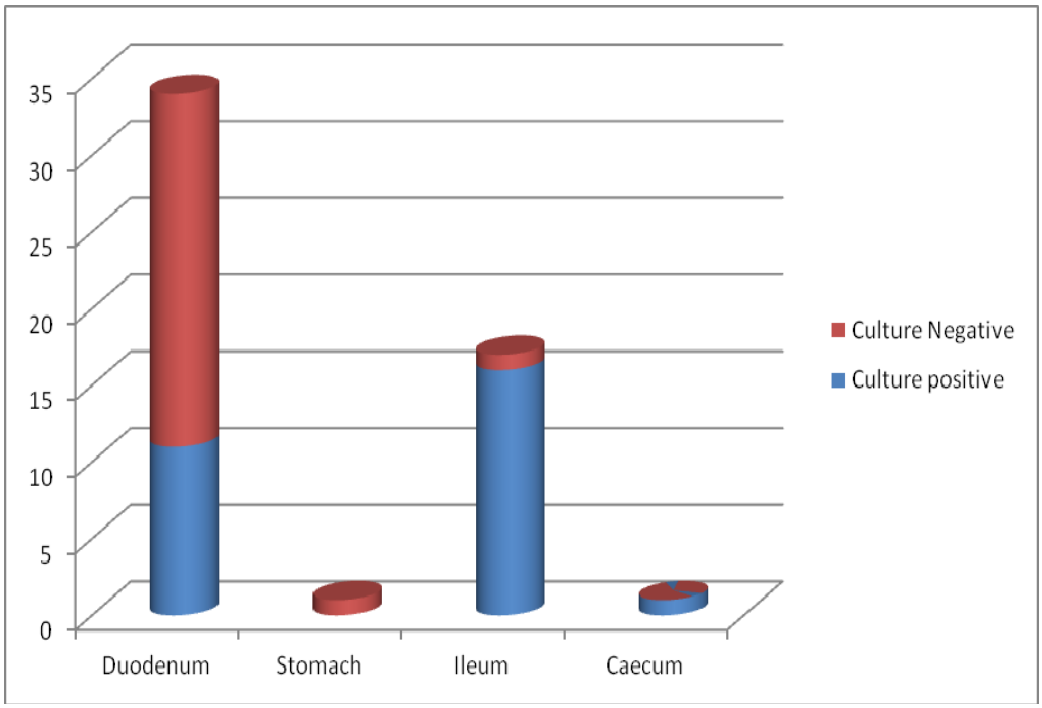


Given below is the list of micro-organisms cultured and the number of patients the micro-organisms are isolated from.



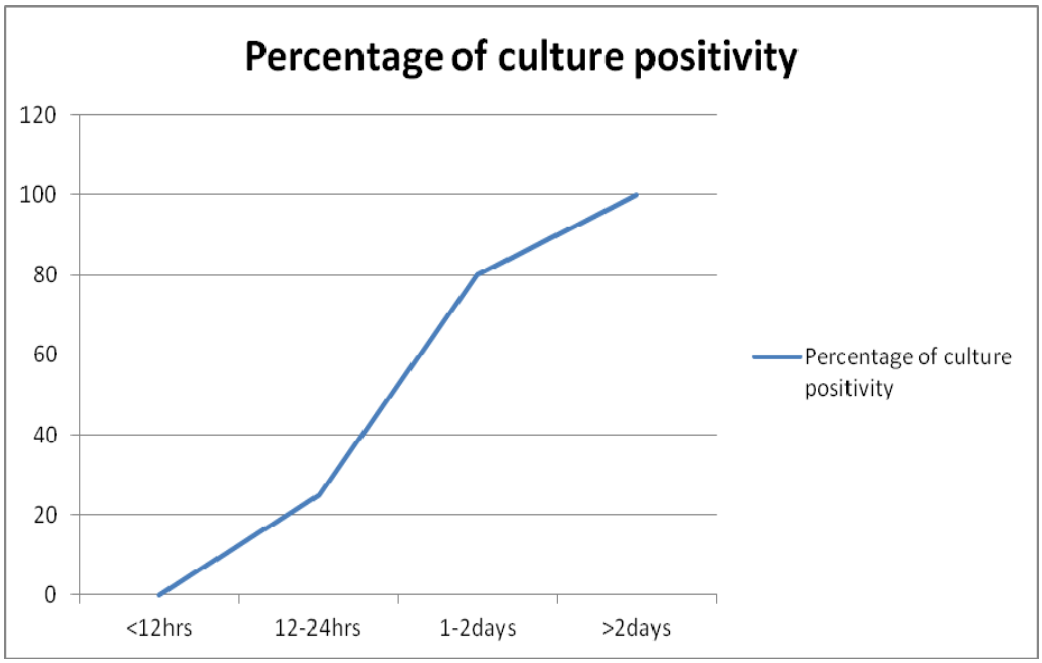
SITE OF PERFORATION & CULTURE POSITIVITY

More proximal the perforation site is, greater will be the possibility of culture negativity. Out of 36 duodenal perforations only 12 cases provided positive culture (33.3%). On the other hand 17 out of 18 ileal perforations yielded a positive culture (94.4%). This is primarily because the proximal GI tract is usually sterile until bacterial translocation sets in, while the terminal ileum is teaming with microbial flora.



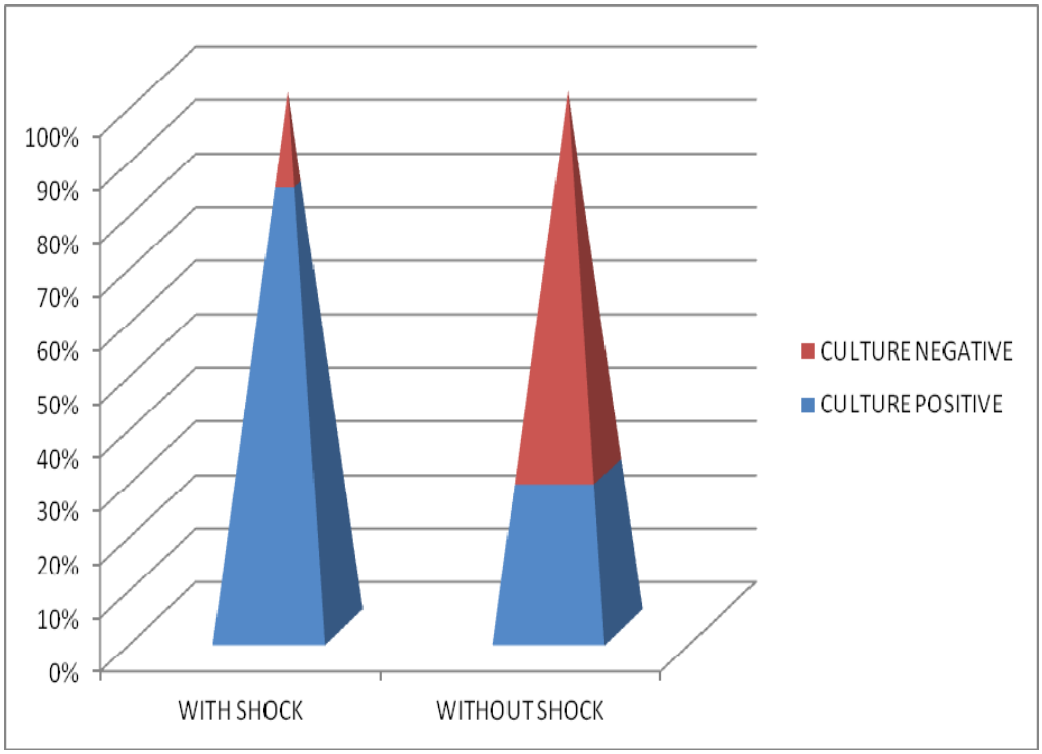
DURATION & CULTURE POSITIVITY

Earlier the presentation of the patient, lesser is the chance of microbial isolate from the peritoneal fluid because the secondary infection has not set in. Out of all 10 cases that presented within 12hrs none had a positive culture. This could be either due to dilution of the perforation fluid by the reactionary fluid from the peritoneum or because the secondary bacterial contamination has not yet occurred. On the other hand 4 out of 16 cases (25%) showed positive culture in patients presenting within 12-24hrs. In patients presenting within 1-2days 12 out of 15 (80%) showed positive culture. On the other hand all the 13 patients presenting later than 2 days showed a positive culture.



SHOCK AND CULTURE POSITIVITY

Presence of shock correlates with the positive culture of peritoneal fluid indicating that the organisms causing shock are from the perforative peritoneal fluid. Out of 54 patients with perforative peritonitis, 23 presented with shock (systolic BP <90mm Hg). The percentage of patients presenting with shock is 42.6%. Of the 23 patients presenting with shock 19 had a positive peritoneal fluid culture (82.6%). In contrast only 9 of 31 patients presenting without shock had a positive peritoneal fluid culture (29%).

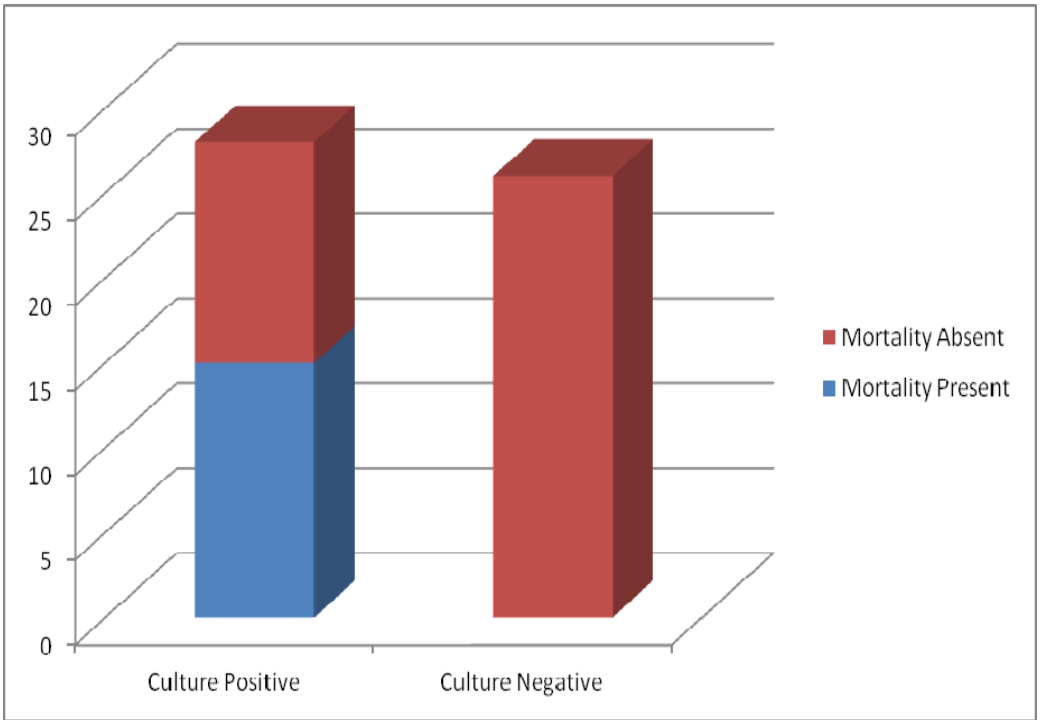


Using Fischer's exact test for a 2x2 contingency table, the two tailed P value is <0.0001 which is statistically significant, which means the presence of organisms in the culture of peritoneal fluid and its correlation with presence of shock is significant.

	Culture positive	**Culture Negative**	**Total**
Shock present	19	4	**23**
Shock absent	9	22	**31**
Total	**28**	**26**	**54**

MORTALITY & CULTURE POSITIVITY

The post-operative mortality of the patient correlates closely with the isolation of organisms from the peritoneal fluid of the perforation peritonitis patients. Out of all54 patients who underwent emergency laparotomy for perforation peritonitis, 15 died in the post-operative period (27.8%). Out of 28 patients who had a positive culture 15 died. In other words, all the patients who died in the post-operative period had a positive peritoneal fluid culture. On the other hand, out of 39 patients who survived, 13 had a positive culture (33.3%) and 26 had a negative fluid culture (66.6%).



Using Fischer's exact test, the two tailed P value was calculated for the following 2x2 contingency table.

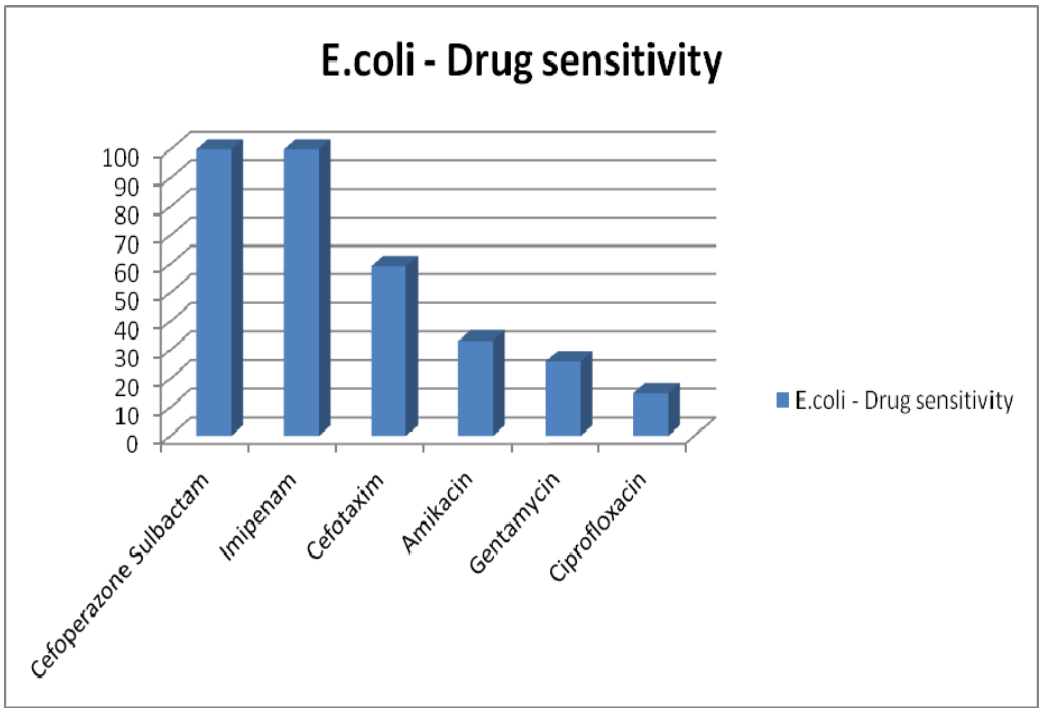
	Mortality present	**Mortality absent**	**Total**
Culture positive	15	13	**28**
Culture negative	0	26	**26**
Total	**15**	**39**	**54**

The P value is <0.0001 using Fischer's exact test, which is statistically very significant meaning that the isolation of bacteria from the peritoneal fluid correlates with the mortality of the perforative peritonitis patients.

DRUG SENSITIVITY PATTERN

1. E.COLI

Out of all 27 cultures from which E.coli was isolated, all were uniformly sensitive to Cefoperazone – Sulbactam combination and Imipenam. 16 isolates showed sensitivity to Cefotaxim sodium (59.26%). 9 isolates showed sensitivity to Amikacin (33.3%). 7 isolates showed sensitivity to Gentamycin (25.9%). Wide spread resistance to ciprofloxacin is seen with only 4 isolates showing sensitivity to the drug (14.8%).



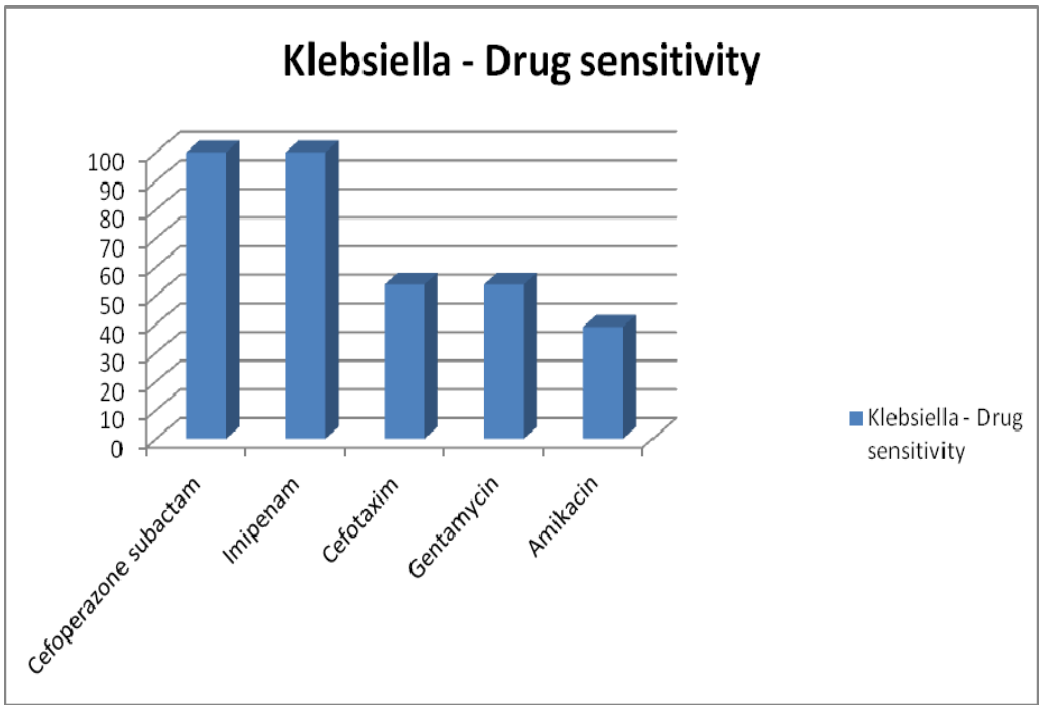
2. BACTEROIDES FRAGILIS

These are gram negative capsulated organisms and are facultative anaerobes. These organisms are consistently sensitive to i.v metronidazole. Of all the 20 isolates from the peritoneal fluid, all showed sensitivity to metronidazole (100%).

3. KLEBSIELLA SPECIES

These are capsulated, non-motile, gram negative bacilli present ubiquitously in the GIT and urinary tract. Of all 13 isolates, all were uniformly sensitive to Cefoperazone – sulbactam and Imipenam. 7 isolates were sensitive to cefotaxim sodium (53.8%). 9 isolates were

sensitive to Amikacin (39.1%). 7 isolates were sensitive to gentamycin (53.8%).



4. ENTEROCOCCUS SPECIES

These organisms commonly cause tertiary peritonitis as these are commonly from gastrointestinal tract. These are one of the common isolates from the small bowel. These organisms are inherently resistant to fluoroquinolones such as ciprofloxacin. Third generation cephalosporins and carbapenams continue to show action against these organisms. All the 4 isolates from the peritoneal fluid are sensitive to cefoperazone – sulbactam and imipenam.

5. PSEUDOMONAS

These are unusual organisms in perforative peritonitis. They are commonly isolated from peritonitis in peritoneal dialysis patients. Of the 3 isolates, all showed sensitivity to cefoperazone – sulbactam and imipenam. 2 isolates showed sensitivity to gentamycin (66.6%) and 1 showed sensitivity to ceftriaxone (33.3%).

6. STREPTOCOCCUS FECALIS

These organisms are gram positive cocci. They are facultative anaerobes commonly found in the intestines. Along with E.coli these organisms form one of the most common isolates from the small bowel. These organisms retain their sensitivity to the common antibiotics such as ampicillin. Of the 2 isolates, all were sensitive to ampicillin, cotrimoxazole, cefoperazone – sulbactam and imipenam.

7. SALMONELLA

Salmonella commonly causes gastroenteritis and typhoid fever. Salmonella is rarely associated with perforative peritonitis. Reports of Salmonella causing spontaneous bacterial peritonitis are present. The 1 isolate showed sensitivity to cefoperazone – sulbactam, imipenam and cefotaxim sodium.

8. ACINETOBACTER

These are gram negative cocci that occur in pairs. These are uniformly aerobic and are non-motile. These are uncommon in community acquired infections. They are commonly nosocomial and in perforative peritonitis it is rare. These bacteria are commonly isolated from the peritoneal fluid of continuous ambulatory peritoneal dialysis patients. Drug resistance is very common in this group. The 1 isolate in peritoneal fluid is sensitive only to imipenam.

Overall almost all isolates except B.fragilis are sensitive to cefoperazone – sulbactam and imipenam. B. fragilis is uniformly sensitive to metronidazole. Hence a combination antibiotic regime should be followed to treat the perforative peritonitis patients. Cefoperazone – sulbactam along with metronidazole can be used as effective empirical antibiotics. Amikacin or Gentamycin can be used as effective adjuncts. Carbapenams can be used as a reserve drug for complicated cases. Since reports of carbapenams resistant strains are fast coming up, isolated use of carbapenams should be strongly discouraged. Carbapenams should be used in combination with other drugs to avoid the development of resistance. Use of carbapenams with β lactamase inhibitor should be encouraged.

CONCLUSION

- Males are more commonly affected by perforative peritonitis than females in the ratio of 4:1.
- Adults in the age group of 25 – 40 are most commonly afftected by perforative peritonitis.
- More proximal the perforation is, lesser is the chance of isolation of organisms from the peritoneal fluid.
- The probability of getting a positive peritoneal fluid culture increases with increase in duration of presenting symptoms.
- A positive perforative peritoneal fluid culture correlates with the development of complications such as shock ($P < 0.0001$).
- A positive perforative peritoneal fluid culture can be used as a positive predicator for post-operative mortality of the patient from septicemia ($P < 0.0001$).

- The infection is almost always polymicrobial. Escherichia coli is the most common single organism isolated followed by Bacteroides fragilis.
- Third generation broad spectrum cephalosporins with β lactamase inhibitor such as Cephaperazone – sulbactam, in combination with Metronidazole act as good empirical antibiotics.
- Amikacin or gentamycin can be used as good adjuncts to increase efficacy, provided the renal parameters are within normal limits.
- Use of Carbapenams can be restricted for complicated cases to avoid emergence of resistance.

GUIDE TO MASTER CHART

1. AUD – Air under diaphragm
2. SITE – Site of perforation
3. D – Duodenum
4. I – Ileum
5. S – Stomach
6. C – Caecum
7. T – Cefotaxim sodium
8. CS – Cefoperazone sulbactam
9. MER – Meropenam
10. M – Metronidazole
11. AK – Amiacin
12. AMP – Ampicillin
13. SEP – Co-trimoxazole
14. GM – Gentamycin
15. CIP - Ciprofloxacin
16. IMP – Imipenam
17. EC – E.coli
18. BF – Bacteroides fragilis
19. E – Enterococci
20. SF – Streptococcus fecalis

PROFORMA

PRE OP : -

Name :

Age :

Sex :

IP No. :

Duration of symptoms :

Co-morbid illness :

DM : yes / no IHD / CAD : yes / no

HT : yes / no

TB : yes / no

<u>On admission :</u>

Vitals :: Pulse : BP :

Anemia : yes / no Icterus : yes / no Pedal edema : yes / no

<u>Investigations :</u>

Hb% :

Air under diaphragm : yes / no

Nature of peritoneal fluid aspirate : Turbid / purulent / feculent / blood

INTRA OP : -

Site of perforation :

Fecal contamination : yes / no

POST OP : -

Organism(s) cultured :

Mortality : yes / no

Drug(s) sensitivity :

Highly sensitive :

Intermediate sensitivity :

Resistant :

BIBLIOGRAPHY

1. Dorairajan LN, Gupta S, Deo SVS, Chumber S, Sharma L. Peritonitis in India-A decades experience. Tropical Gastroenterology. 1995;16:33–38.

2. Sharma L, Gupta S, Soin AS, Sikora S, Kapoor V. Generalised peritonitis in India-The tropical spectrum. Jap J Surg. 1991;21:272–77. doi: 10.1007/BF02470946.

3. Suanes C, Salvesan H, Espehang B. A multifactorial analysis of factors related to lethality after treatment of perforated gastrduodenal ulcer. Ann Surg. 1989;209:418–23.

4. Washington BC, Villalba MR, Lauter CB. Cefamendole-erythromycin-heparin peritoneal irrigation. An adjunct to the surgical treatment of diffuse bacterial peritonitis. Surgery.1983;94:576–81.

5. Nomikos IN, Katsouyanni K, Papaioannou AN. Washing with or without chloremphenicol in the treatment of peritonitis. A prospective clinical trial. Surgery. 1986;99:20–25.

6. Shinagawa N, Muramoto M, Sakurai S, Fukui T, Hon K, Taniguchi M, Mashita K, Mizuno A, Yura J. A bacteriological study of perforated duodenal ulcer. Jap J Surg. 1991;21:17.

7. Khanna AK, Mishra MK. Typhoid perforation of the gut. Postgraduate Medical Journal.1984;60:523.

8. Noon GP, Beall AC, Jorden GL. Clinical evaluation of peritoneal irrigation with antibiotic solution. Surgery.

9. Bose SM, Kumar A, Chaudhary A, Dhara I, Gupta NM, Khanna SK. Factors affecting mortality in small intestinal perforation. Indian Journal of Gastroenterology.

10. Crawfurd E, Ellis H. Generalised peritonitis-The changing spectrum. A report of 100 consecutive cases. British Journal of Clinical Practice 1985

11. People TB. Candida with perforated peptic ulcer. Surgery. 1986;100:758–64.

12. Bohen J, Boulanger M, Meakins L. Prognosis in generalized peritonitis. Arch Surg. 1983;118:285.

13. Nadkarni KM, Shetty SD, Kagzi RS, Pinto AC, Bhalerao RA. A small bowel perforation- A study of 32 cases. Arch Surg. 1981;116:53–57.

14. Ellis H. Incisions, closures and management of the wound. In: Zinner MJ, Schwartz IS, Ellis H, editor. Maingot's Abdominal operations.

15. Keane WF, Vas SI. Peritonitis. In: Gokal R, Nolph KD (eds). The Textbook of Peritoneal Dialysis. Boston: Kluwer, 1994, pp. 473-502.

16. Cruse PJE, Foord R. A five-year prospective study of 23,649 surgical wounds. Arch Surg 1973

17. Farha GF, Chang FC, Matthews EH. Drainage in elective cholecystectomy. Am J Surg 1981

18. Johnson G, Gilsdorf R. Routine versus selective drainage of the gallbladder bed after cholecystectomy. American Journal of Surgery

19. Hunt JL. Generalized peritonitis: to irrigate or not to irrigate the abdominal cavity.

20. Dawson JL. A study of some factors affecting the mortality rate in diffuse peritonitis.

NO	NAME	AGE	SEX	PRIMARY SYMPTOM	DURATION	FEVER	AUD	ASPIRATE NATURE	SHOCK	SITE	ANTIBIOTICS GIVEN
1	HARISH	29	M	ABDOMEN PAIN	12hrs	N	Y	TURBID	N	D	T,M
2	MATHUSUDHAN	54	M	ABDOMEN PAIN, DISTENSION	2days	Y	Y	PURULENT	Y	I	CS,M
3	SUNDAR	32	M	ABDOMEN PAIN	1day	Y	Y	TURBID	N	D	Mer,M
4	DINESH	30	M	ABDOMEN PAIN	1day	N	Y	TURBID	N	D	Mer,M
5	ARUMUGAM	43	M	ABDOMEN PAIN	2days	Y	Y	BILIOUS	Y	D	Mer,M
6	KUMAR	38	M	ABDOMEN PAIN, DISTENSION	1day	Y	Y	TURBID	N	D	CS,M
7	KALAIARASI	36	F	ABDOMEN PAIN	2days	Y	Y	TURBID	N	I	Mer,M
8	ELUMALAI	68	M	ABDOMEN PAIN	12hrs	Y	Y	TURBID	N	I	CS,M
9	VANAJA	32	F	ABDOMEN PAIN	6hrs	N	Y	BILIOUS	Y	D	T,M
10	GOUSE BASHA	46	M	ABDOMEN PAIN, CONSTIPATION	3days	Y	Y	FECULENT	N	I	Mer,M
11	THANGAVELU	35	M	ABDOMEN PAIN, DISTENSION	3days	Y	Y	FECULENT	Y	I	Mer,M
12	ARULRAJ	44	M	ABDOMEN PAIN	2days	Y	Y	BILIOUS	Y	D	Mer,M
13	RAMADOSS	36	M	ABDOMEN PAIN	12hrs	N	Y	TURBID	N	D	Mer,M
14	SATHISH	24	M	ABDOMEN PAIN, DISTENSION	1day	N	Y	TURBID	N	D	CS,M
15	GOVINDHAN	33	M	ABDOMEN PAIN	1day	Y	Y	PURULENT	N	I	Mer,M
16	KUMARESAN	31	M	ABDOMEN PAIN	2days	N	Y	TURBID	N	D	T,M,AK
17	KALAISELVI	42	F	ABDOMEN PAIN, DISTENSION	3days	Y	Y	BILIOUS	Y	D	Mer,M
18	MITUN GUPTA	37	M	ABDOMEN PAIN	1day	Y	Y	TURBID	N	D	CS,M
19	ELUMALAI	50	M	ABDOMEN PAIN	2days	N	Y	TURBID	Y	D	CS,M
20	UMAPATHY	54	M	FEVER, CONSTIPATION, DISTENSION	4days	Y	N	FECULENT	Y	I	Mer,M
21	CHELLAPANDI	36	M	ABDOMEN PAIN	2days	N	Y	BILIOUS	N	D	Mer,M
22	RAMALINGAM	61	M	ABDOMEN PAIN	3days	Y	Y	FECULENT	Y	I	Mer,M
23	GOVINDARAJ	43	M	ABDOMEN PAIN	2days	Y	N	TURBID	N	I	Mer,M
24	KRISHNASAMY	33	M	ABDOMEN PAIN	1day	Y	Y	TURBID	N	D	Mer,M
25	DEVEDHANAM	32	F	ABDOMEN PAIN	2days	Y	Y	BILIOUS	Y	D	Mer,M
26	ELANGOVAN	56	M	ABDOMEN PAIN	1day	N	Y	TURBID	N	D	T,M

27	MARIAM SUSAI	59	M	ABDOMEN PAIN, CONSTIPATION	4days	Y	N	TURBID	Y	C	Mer,M
28	MAHESH	45	M	ABDOMEN PAIN	1day	N	Y	TURBID	Y	D	CS,M
29	CHELLADURAI	52	M	ABDOMEN PAIN	12hrs	N	N	TURBID	N	D	T,M
30	PARIMALAM	34	F	ABDOMEN PAIN, DISTENSION	3days	Y	Y	TURBID	Y	I	Mer,M
31	RAJENDRAN	28	M	ABDOMEN PAIN	1day	N	Y	BILIOUS	N	D	Mer,M
32	RAJA	46	M	ABDOMEN PAIN	2days	Y	Y	BILIOUS	Y	D	CS,M
33	DURAI	43	M	ABDOMEN PAIN	4days	Y	Y	TURBID	Y	I	Mer,M
34	AROKIYASAMY	58	M	ABDOMEN PAIN	1day	N	Y	TURBID	N	S	Mer,M
35	GOVINDASAMY	36	M	ABDOMEN PAIN	2days	Y	Y	BILIOUS	N	D	T,M
36	VASANTH	28	M	ABDOMEN PAIN, FEVER	3days	Y	Y	TURBID	Y	I	Mer,M
37	SUNDARESAN	42	M	ABDOMEN PAIN	4days	Y	Y	FECULENT	Y	I	Mer,M
38	VISALATCHI	38	F	ABDOMEN PAIN	12hrs	N	Y	TURBID	N	D	CS,M
39	MURUGESAN	33	M	ABDOMEN PAIN	2days	N	Y	BILIOUS	Y	D	Mer,M
40	GAJALAKSHMI	43	F	ABDOMEN PAIN, DISTENSION	2days	N	Y	BILIOUS	Y	D	Mer,M
41	ANSAR BASHA	38	M	ABDOMEN PAIN	12hrs	N	Y	TURBID	N	D	T,M
42	PANDIYAN	44	M	ABDOMEN PAIN	1day	Y	Y	TURBID	N	I	T,M
43	LAKSHMANAN	29	M	ABDOMEN PAIN	1day	Y	Y	TURBID	N	D	Mer,M
44	APPU	31	M	ABDOMEN PAIN	2days	Y	Y	BILIOUS	N	D	Mer,M
45	THARUN	33	M	ABDOMEN PAIN, DISTENSION	2days	Y	Y	BILIOUS	Y	D	CS,M
46	KANAGA	38	F	ABDOMEN PAIN	1day	Y	Y	TURBID	N	I	Mer,M
47	MOHANKUMAR	54	M	ABDOMEN PAIN	12hrs	N	Y	TURBID	N	D	T,M
48	ARUMUGAM	32	M	ABDOMEN PAIN	3days	Y	Y	BILIOUS	Y	D	Mer,M
49	PRABAKAR	66	M	ABDOMEN PAIN	3days	Y	Y	TURBID	Y	I	Mer,M
50	YESU MAGAN	28	M	ABDOMEN PAIN	1day	N	Y	TURBID	N	D	CS,M
51	CHINNAPONNU	44	F	ABDOMEN PAIN, DISTENSION	1day	N	N	BILIOUS	N	D	T,M
52	VELAMMAL	32	F	ABDOMEN PAIN, DISTENSION	6hrs	N	Y	TURBID	N	D	T,M
53	MURUGAN	28	M	ABDOMEN PAIN	12hrs	N	Y	TURBID	N	D	Mer,M
54	CHARUMATHY	44	F	FEVER, CONSTIPATION, DISTENSION	3days	Y	Y	FECULENT	Y	I	Mer,M

MORT	ORGANISMS CULTURED	DRUG SENSITIVITY PATTERN							
		EC	BACT	KLEB	ENT	PSEUD	S.FECALIS	SALM	ACINET
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	EC, K, BF	CS, AK, CIP, IMP	M	CS, T, AK, IMP	Nil	Nil	Nil	Nil	Nil
P	EC, BF, P	CS, AK, T, IMP	M	Nil	Nil	CS, IMP	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	EC, K	CS, T, IMP	Nil	CS, AK, T, IMP	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
P	EC, BF	CS, T, AK, GM, IMP	M	Nil	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
P	EC, K, BF, E	CS, AK, IMP	M	CS, AK, GM, IMP	CS, IMP	Nil	Nil	Nil	Nil
A	EC, K, BF	CS, AK, CIP, IMP	M	CS, T, AK, IMP	Nil	Nil	Nil	Nil	Nil
A	EC, BF	CS, GM, IMP	M	Nil	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	EC, K, A	CS, T, IMP	Nil	CS, T, GM, IMP	Nil	Nil	Nil	Nil	IMP
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
P	EC, BF	CS, GM, IMP	M	Nil	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
P	EC, K	CS, T, GM, IMP	Nil	CS, T, AK, IMP	Nil	Nil	Nil	Nil	Nil
P	EC, K, BF, P	CS, GM, IMP	M	CS, GM, IMP	Nil	CS, GM, IMP	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
P	EC, K, BF	CS, T, IMP	M	CS, GM, AK, IMP	Nil	Nil	Nil	Nil	Nil
A	EC, BF	CS, T, IMP	M	Nil	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
P	EC, SF	CS, T, GM, IMP	Nil	Nil	Nil	Nil	AMP, SEP, CS, IMP	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil

A	EC, BF	CS, T, AK, GM, IMP	M	Nil	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
P	E, BF, SF	Nil	M	Nil	CS, T, IMP	Nil	AMP, SEP, CS, IMP	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	EC, K	CS, GM, IMP	Nil	CS, AK, GM, IMP	Nil	Nil	Nil	Nil	Nil
P	EC, BF	CS, AK, IMP	M	Nil	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	E	Nil	Nil	Nil	CS, IMP	Nil	Nil	Nil	Nil
A	EC, BF	CS, T, CIP, IMP	M	Nil	Nil	Nil	Nil	Nil	Nil
P	EC, K, BF,S	CS, T, GM, IMP	M	CS, T, GM, IMP	Nil	Nil	Nil	T, CS, IMP	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
P	EC	CS, AK, GM, IMP	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	EC, K	CS, T, CIP, IMP	Nil	CS, T, AK, GM, IMP	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	EC, BF	CS, T, AK, GM, IMP	M	Nil	Nil	Nil	Nil	Nil	Nil
A	EC	CS, AK, IMP	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	EC, K, BF	CS, AK, GM, IMP	M	CS, IMP	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
P	EC, BF, P	CS, T, AK, IMP	M	Nil	Nil	CS, GM, Tr, IMP	Nil	Nil	Nil
P	EC, BF	CS, T, AK, IMP	M	Nil	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
A	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
P	EC, K, BF, E	CS, AK, GM, T, IMP	M	CS, AK, IMP	CS, IMP	Nil	Nil	Nil	Nil